A CLINICAL STUDY OF ABDOMINAL WOUND DEHISCENCE

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In

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DEPARTMENT OF GENERAL SURGERY

TIRUNELVELI MEDICAL COLLEGE

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Dear, Dr.A.CHUDAR, MBBS., The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 10.03.2017

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1. TIREC Application Form ☐
2. Study Protocol ☐
3. Department Research Committee Approval ☐
4. Patient Information Document and Consent Form in English and Vernacular Language ☐
5. Investigator's Brochure ☐
6. Proposed Methods for Patient Accrual Proposed ☐
7. Curriculum Vitae of the Principal Investigator ☐
8. Insurance /Compensation Policy ☐
9. Investigator's Agreement with Sponsor ☐
10. Investigator's Undertaking ☐
11. DCGI/DGFT approval ☐
12. Clinical Trial Agreement (CTA) ☐
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA) ☐
14. Clinical Trials Registry-India (CTRI) Registration ☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
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This is certify that this dissertation work title **"A CLINICAL STUDY OF ABDOMINAL WOUND DEHISCENCE"** of the candidate **Dr. A. CHUDAR** with registration Number **221611353** for the award of **M.S.** Degree in the branch of **GENEARL SURGERY (I).** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **6 percentage** of plagiarism in the dissertation.

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22

INTRODUCTION

Abdominal wound dehiscence, also known as burst abdomen, acute wound failure, wound disruption, evisceration or eventration, remains one of the most dramatic and serious developments confronting the general surgeon. Few postoperative events cause such morbidity, and when accompanied by necrotizing fasciitis, none is as potentially disfiguring.

Abdominal wound dehiscence is defined as the postoperative separation of layers of a laparotomy wound, with or without eventration. Despite major advances in the preoperative care of surgical patients, including the introduction of broader spectrum antibiotics and an improved understanding of the effects of systemic illness on wound healing, the incidence of abdominal wound dehiscence has remained constant at 0.4 to 3.0%.[1, 2]

Two general factors play contributory roles in causing wound dehiscence - metabolic and local anatomic abnormalities and technical factors. Many aspects of the latter are within the surgeon's control, such as the site of the abdominal incision, technique of closure and type of suture employed, the use of retention sutures, and the placement of drains and enterostomies in relation to the wound. Metabolic abnormalities are commonly corrected before elective operations, a factor which increases the risks in emergency operations. At the same time, the

unalterable variables of patient age, the procedure itself - whether it be elective, emergent, clean, or contaminated, and associated systemic illness have been shown to be contributory.[3-7]

Although specific guidelines describe the reoperative management of abdominal wound dehiscence, more important is recognition at initial operation of the patient who is at risk for wound separation as well as implementing at that time measures to prevent its occurrence.

AIMS AND OBJECTIVES

To study the clinical profile of patients with the diagnosis of abdominal wound dehiscence with respect to:

- age distribution
- gender incidence
- clinical presentation
- nature of preceding surgery (elective or emergency)
- contributing factors - local (type of incision, suture material used for abdominal closure) and systemic (anemia, hypoproteinemia, postoperative infection, postoperative pulmonary complications, obesity, comorbid conditions, drug use)
- management
- outcome

REVIEW OF LITERATURE

SURGICAL ANATOMY OF ABDOMINAL WALL[7]

The abdominal wall is a complex musculoaponeurotic structure that is attached to the vertebral column posteriorly, the ribs superiorly, and the pelvic bones below. The abdominal wall is composed of nine layers (Fig.1). From without in, they are:

(1) Skin

(2) Tela subcutanea (subcutaneous tissue)

(3) Superficial fascia (Scarpa fascia)

(4) External abdominal oblique muscle

(5) Internal abdominal oblique muscle

(6) Transversus abdominis muscle

(7) Endoabdominal (transversalis) fascia

(8) Extraperitoneal adipose and areolar tissue

(9) Parietal peritoneum

The **rectus muscles** and rectus sheath require special description. The muscles are paired right and left, and they extend from the fifth rib superiorly to the pubis inferiorly. They lie in apposition to each other, separated only by the linea alba. The rectus muscles serve to support the abdominal wall and to flex the vertebral column. Each muscle is contained

within a fascial sheath, the **rectus sheath**, which is derived from the aponeuroses of the three flat abdominal muscles.

The relationship of the aponeuroses of the flat muscles is not constant throughout the course of the rectus muscle. The relationship is different above and below the semicircular line of Douglas, which is about halfway between the umbilicus and the pubic symphysis (Fig.2). Above the semicircular line, the rectus sheath is strong posteriorly. Here the posterior sheath is composed of fascia from the internal oblique muscle, the transversus abdominis muscle, and the transversalis fascia. Anteriorly, above the semicircular line, the rectus sheath is composed of the external oblique aponeurosis and the anterior lamella of the internal oblique aponeurosis. Below the semicircular line, which is the point at which the inferior epigastric artery enters the rectus sheath, the posterior rectus sheath is lacking because the fasciae of the flat muscles pass anterior to the rectus muscle. The muscle, below the semicircular line, is covered posteriorly by a thin layer of transversalis fascia, which is usually transparent when viewed from the inside at operation. The rectus abdominis muscles are held close together near the anterior midline by the linea alba. The **linea alba** itself has an elongated triangular shape and is based at the xiphoid process of the sternum. The linea alba narrows

considerably below the umbilicus, so that the medial edge of one rectus muscle may actually overlap the other.

PHYSIOLOGY OF WOUND HEALING[8]

A wound initially is tissue that has lost normal structure and functions as the result of internal or external forces. Wound healing is the sequence of cellular and molecular events activated at the time of injury resulting in a time-dependent pattern of tissue repair. Classically, the phases of wound healing are described as hemostasis, inflammation, fibroproliferation, and remodeling (maturation).

(a) Hemostasis:

Before a wound can heal it must stop bleeding. Therefore, the earliest phase of wound healing following injury is characterized by the deposition of fibrinogen, a soluble plasma protein synthesized by the liver and secreted into the systemic circulation. Fibrinogen extravasates from disrupted blood vessels and fills the gap of the wound.

(b) Inflammation:

The cellular and humoral inflammatory phase is induced next, and an immune barrier is established against pathologic microorganisms. Necrotic tissue locally releases cellular breakdown products capable of maintaining and amplifying the early inflammatory response following injury. The increased permeability of vessels adjacent to the injury facilitates the migration of inflammatory cells into the wound which leads to phagocytosis of invading

microbes and release of cytochemoattractants. Hence if a wound infection develops, healing will be delayed. Circulating monocytes enter the wound in a second wave of inflammatory cells within 24 hours after the appearance of neutrophils. Monocytes terminally differ into tissue macrophages. Macrophages are vital for clearing the wound of microbes and devitalized tissue, as well as for the production of a new connective tissue matrix.

(c) Fibroproliferation and Remodeling:

Once hemostasis is achieved, ongoing injury has ceased, and an immune barrier is in place, wound healing trajectories shift toward fibroplasia and tissue repair. Scar tissue replaces normal tissue following injury and is often a source of subsequent wound complications. Over time, wound matrix cell number diminishes and collagen bundles are increasingly organized during remodeling. This final phase of wound healing can continue for years until a maximum wound strength plateau is finally reached. In dermal wounds, overlying epidermal cells begin to migrate across the tissue defect at about this time to restore the skin's epithelial barrier function. Collagen is the major protein component of wound connective tissue. Unwounded dermis contains approximately 80% type I collagen and 20% type Ill collagen. Acute wound granulation tissue, in contrast, expresses twice as much type III collagen. Normal collagen

synthesis and secretion requires hydroxylation of lysine and proline residues. The cofactors necessary for enzymatic collagen hydroxylation are ferrous iron, molecular oxygen, a-ketoglutarate, and vitamin C. Impaired wound healing results from deficiencies in any of these cofactors, as during tissue hypoxia or with diets low in vitamin C. In acute wound granulation tissue mature collagen fibers are oriented in overlapping arrays parallel to the wound surface and usually along lines of maximum tension. When the wound defect is filled, the maturing granulation tissue undergoes remodeling. The density of macrophages and fibroblasts is reduced. There is also no regeneration of lost sub epidermal appendages such as hair follicles or sweat glands following skin healing.

The Lag Phase

The ―lag phase‖ of wound healing is defined as the earliest period of time following wounding when hemostasis, inflammation, and early fibroplasias are induced. It is during the lag phase of wound healing that acute wounds are most vulnerable to mechanical failure (dehiscence). The wound tensile strength is 0% to 30% of its maximum value during the first 7 days following wounding.

Wound failure occurs when there is an abnormality in the magnitude or duration of the sequential components of tissue repair. Inadequate hemostasis due to platelet dysfunction or poor technique results in

hematoma formation with ensuing mechanical disruption of the provisional wound matrix. Delayed or deficient inflammatory responses increase the risk of wound contamination or infection. A prolonged inflammatory response due to foreign material delays the progression of tissue repair into the fibroproliferative phase in which rapid gains in breaking strength and wound contraction should occur. Impaired fibroblast activation in turn impedes the establishment of the early wound matrix and synthesis of immature scar. Epithelialization requires an underlying functional bed of granulation tissue. Obstacles to normal wound healing therefore shift the wound healing trajectory and result in wound complications.

EPIDEMIOLOGY

SCOPE OF THE PROBLEM: INCIDENCE

Despite major advances in the perioperative care of surgical patients, the incidence of abdominal wound dehiscence has remained constant at 0.4 to 3.0%.[1, 2]

The comprehensive literature review by Poole[9] in 1985 investigating the incidence of AWD encompassed some 320,000 abdominal operations performed during the past 35 years. Overall there were approximately 1900 dehiscences, yielding a mean incidence of 0.59%. Poole and others, however, are critical of the accuracy of this low rate, citing as an explanation the inclusion of muscle-splitting appendicectomy incisions, herniorhaphies, and gynaecologic procedures in many of the trials. The incidence of AWD with these incisions is sufficiently low that their inclusion artificially lowers the incidence realized in major abdominal surgery. Based on the above observation, Poole proposed that a more accurate estimate in general abdominal surgery was 1 to 3%. To avoid ambiguity, we define abdominal wound dehiscence as the postoperative separation of all three layers of a laparotomy wound, with or without eventration.

Table 1: Incidence of AWD in various clinical trials:

Author	Year	No. of patients	Dehiscences (%)	Primary variable
Irvin et al[10]	1977	200	2(1.0)	Closure(layered vs. mass)
Greenall et al[1]	1980	577	2(0.4)	Incision(midline vs. transverse)
Guillou[11]	1980	207	1(0.5)	Incision(midline vs. paramedian)
Corman et al[12]	1981	161	1(0.6)	Suture
Bucknall and Ellis[13]	1981	210	2(0.95)	Suture
Donaldson[14]	1982	231	0(0)	Suture
Richards et al[15]	1983	571	8(1.0)	Closure(interrupted vs. continuous)
Gammelgaard and Jensen[16]	1983	306	1(0.33)	Suture
Ellis et al[17]	1984	175	1(0.6)	Incision(transverse vs. vertical)
Fagniez et al[18]	1985	3135	58(1.8)	Closure(interrupted vs. continuous)

The incidence of burst abdomen in India has been reported to be 5-7%.

RISK FACTORS

DEFINING THE RISK : THE PATIENT

The seeds of wound disruption may be present before the patient goes to the

operating room. - W. I Wolff

Patient related risk factors play an important role in the development of AWD. Advancing age, poor vascular supply, male sex, preexisting pulmonary disease, malnutrition and immunosuppression are the most significant implicated factors today.

Advanced **age** is the single factor consistently emphasized by most authors that coincides with a decreased ability to fight off infection. Wolff found the incidence of wound disruption in patients aged >45 years to be 4 times that in the younger age group (5.4% vs. 1.3%)[3]. Similar results were noticed by McCallum and Link[19], who demonstrated a threefold increase in incidence in the older population (4.5% vs. 1.6%). The high incidence of AWD noticed in the elderly is explained by a diminished rate of cell proliferation which has a detrimental impact on healing process.

Male **gender** is an additional predisposing factor emphasized in most reviews. The ratio of males to females experiencing this complication ranges from 1.6:1 to 3:1. The male predisposition to wound failure may be related to the more relaxed

abdominal wall in females following pregnancy, greater postoperative physical activity in males, and a higher incidence of preoperative pulmonary disorders in males resulting in excessive postoperative cough. However, with the percentage of women smokers increasing each year, a shift in these statistics is likely. All of these factors tend to increase wound tension.

Preoperative **pulmonary disease** and subsequent postoperative respiratory complications have a well-defined role in the development of wound failure. Wolff reported severe paroxysmal coughing prior to wound disruption in over 60% of cases[3]. Alexander and Pavdden[20] and Hampton[21] also noted pulmonary complications to be the most frequent event leading to postoperative wound disruption. With preoperative use of bronchodilator agents, elimination of tobacco, treatment of bronchitis, and respiratory therapy, optimal pulmonary function is achieved. Studies have shown that cessation from smoking for as little as one week preoperatively can lessen patients' morbidity postoperatively and a diminished incidence of postoperative wound failure.

Research implicating adequate **nutrition** in achieving secure surgical wounds began 50 years ago with the important observations of Thompson et al[22] and Elman[23], that hypoproteinemia leads to greatly impaired wound healing. Kraybill documented hypoproteinemia in 6 of 7 patients

experiencing postoperative wound disruption[24], and reports of the association have continued ever since. While protein deficiency is rare in the United States today, its prevalence in developing countries and occurrence in association with other diseases remains a continuing problem. Reports by Wolff[3], Alexander and Pavdden[20] and Keill[25] that 62%, 71% and 85% of their respective wound dehiscences were associated with hypoproteinemia emphasize the magnitude of the problem. Clearly every effort should be made to correct these protein deficiency states before elective surgery. Continued improvements in both enteral and parenteral formulas available today have made this feasible in even the most sever forms of malnourishment.

THE NATURE OF THE OPERATION

Emergency laparotomy (irrespective of the organ system involved), gastric operations (particularly for peptic ulcer disease and haemorrhage), and procedures involving the small and large bowel have increased rates of dehiscence.

The rate of dehiscence following procedures on stomach, small and large bowel was found to be twice that after operations on the biliary tree[26]. Whether anticipated or unexpected, the single common factor involves some degree of peritoneal contamination and its subsequent adverse impact on the abdominal wound[6,25]. Emergent laparotomy predisposes to subsequent wound problems, though the reason for wound

failure is not well defined. Penninckx et al reported an incidence of dehiscence in emergent laparotomies of 6.7%, more than two-and- a-half times that observed in elective cases[27]. This parallels the incidence reported by Mendoza et al of 6.2% during emergent gastroduodenal surgery for haemorrhage[28]. Presumably, similar factors are operative in these emergent cases as in those involving the GIT, particularly some degree of peritoneal contamination. These emergencies may be associated with a break in technique as a result of hurried operation in an unstable patient or, as more frequently is the case, they may involve a procedure on an open unprepared bowel. One must also take into account the mechanism of the injury leading to the emergent laparotomy. Regardless of the cause, the common factor is wound contamination, and unless extra preventive measures are undertaken, dehiscence on the basis of infection may ensue.

Carcinoma leads to cachexia and marked anemia due to anorexia, haemorrhage and bone marrow depression. The effect of cancer on wound healing was studied by Wyatt et al[68], who found that, although wound healing may proceed in a relatively unimpeded manner for many patients with cancer, there is a potential for wound failure due to the nature and effects of the oncologic disease process and its treatments.

While pulmonary function should be optimized and nutritional deficiencies corrected before elective surgery, little can be done preoperatively in the emergent situation. Similarly, advancing age and

male sex, as well as the requirement for an emergency procedure or one involving the gastrointestinal tract, are unalterable variables. Although the presence of one of these alone may be relatively harmless, their presence in combination should be considered an indication for measures to consider dehiscence a realistic possibility.

LOCAL FACTORS CONTRIBUTING TO DEHISCENCE: THE INCISION

Deciding the most appropriate incision for a given patient is based a variety of factors. Considerations, in order of importance, include

(1) Access afforded;

(2) Expediency of entry and closure (particularly in the unstable trauma patient);

(3) Relative postoperative pain and pulmonary complications (especially in the face of chronic pulmonary disease); and

(4) In the presence of multiple risk factors, the associated incidence of postoperative wound disruption and incisional hernia formation.

The major controversy during the past 50 years has involved transverse versus vertical abdominal incisions.

The musculoaponeurotic fibers of all three layers of the abdominal wall run in a predominantly transverse direction. Active contraction of the oblique and transversus musculature results in forces that are directed

laterally away from the midline. These forces run perpendicular and in opposing directions on either side of a vertical incision, thereby tending to distract the wound edges. Transverse incisions are affected little by these forces.

Sloan demonstrated a 30-fold increase in wound tension in vertical as opposed to transverse incisions in lightly anesthetized patients[29]. Subsequent publications by Hampton, McCallum, and Lehman and Partington also cited excessive disruption rates with the vertical wound[19,21,30]. Simultaneously several authors documented less postoperative pain and concomitantly fewer postoperative pulmonary complications when transverse incisions were used. The resurgence of vertical incision popularity may be related to the exposure they provide when aided by the new fixed retractors. In the face of trauma, the expediency with which the abdomen may be opened and closed is of primary importance, and continuing civilian abdominal trauma demands this attribute of the vertical midline incision. More relevant to the persistent popularity of the midline incision is the realization by many that its use does not necessarily imply a sacrifice in terms of wound security.

From a strictly anatomic viewpoint, Tera and Aberg, using human cadavers, demonstrated a clear superiority in the holding power of the midline incision over the transverse when sutures were placed lateral to the transition between the linea alba and rectus sheath (22.9 kPa vs. 13.3

kPa)[31]. Similarly, Leaper et al also found the midline aponeurosis in human cadavers to have the greatest suture-holding capacity[32]. Finally, Higgins et al used a rabbit model to demonstrate that a vertical incision closed with the Smead-Jones technique was far stronger at 7 days than a transverse incision closed with the same suture technique[33].

Currently, there is little evidence that incision orientation alone plays a significant role in the etiology of abdominal wound dehiscence. The nature of the operation itself, its attendant risk of wound infection, the technique of closure, and certain postoperative factors are more critical.

INTRAOPERATIVE PREVENTION

Protecting the Wound

The time to think about and prepare for a possible wound separation is prior to and during operation, not after disruption has occurred.

- Lehman and Partington

Once the peritoneal cavity has been opened, every effort should be made to prevent contamination of the wound with potential pathogens. This is frequently more difficult in a reoperation than in a primary procedure. Since dehiscence occurs far less frequently than wound sepsis, and often in its absence, it is difficult extrapolate from wound infection to a specific incidence of dehiscence. Suffice it to point out that wound infections were noted in 72% of dehiscences reviewed by Keill et al, whereas the control population of intact wounds had a 3% infection rate[25]. Thus, while local mechanical factors contribute heavily to subsequent wound disruption, the effect of infection on tissue strength cannot be underestimated.

The high dehiscence rates following gastric operations or those involving large or small intestine were described earlier. The relationship between intraoperative contamination of the peritoneal cavity and subsequent wound infection is well established and continues to be significant[34,35,36].

Any violation of the gastrointestinal tract, be it iatrogenic, for decompressive purposes, or for resection of gangrenous bowel, will be accompanied by significant wound contamination, and if skin is closed, the risk of incisional infection is increased. Raahave et al have quantitatively defined this risk by demonstrating an exponential relation ship between intraoperative bacterial density within the wound and subsequent wound infection[37]. The critical ―infective dose‖ observed in that trial was 4.6 x 10^5 colony-forming units per square centimeter (CFU/cm^2).

In an attempt to diminish intraoperative wound involvement during contaminated procedures, multiple different impermeable skin and wound drapes have been introduced. Most prospective trials have been unable to demonstrate a significant reduction in wound infections with their use[38,39]. Nonetheless, the principle of avoiding wound and generalized peritoneal contamination by isolating the pathologic area of the intestine should be recognized as theoretically sound one to be adhered to at all times.

The use of drains in abdominal operations continues to be controversial. Clear indications in the past have included[40]:

(1) anticipated leakage from an adjacent organ such as the pancreatic or gallbladder bed

(2) isolated abscess cavities requiring drainage to achieve collapse and progressive healing from the deepest portion outward

(3) a worrisome anastomosis as a result of tension or compromised tissue at the suture line.

The currently accepted indication for abdominal drainage is a clear recognition that the drain is essential to carry away infected material or digestive enzymes, or other chemically irritating fluid, which will impair wound healing. It is strongly recommended that drainage be performed through a separate, more dependent stab wound in the abdominal wall, well away from the operative incision)

Finally, an enteral stoma, be it from the stomach, small bowel, or colon, should be considered a similar infectious hazard, particularly when the wound is left open. This was emphasized by Wolff, who noted that as many as 8 of 45 wound dehiscences reported were directly attributable to bringing an enteral stoma through the operative incision[3]. This outcome should be anticipated and avoided by extraincisional placement of the stoma. Finally, the prevention of wound contamination during any operation demands continuous attention to meticulous technique. The delicate handling of tissue, removal of foreign material, debridement of necrotic tissue and absolute hemostasis before closure all fall within this realm.

Fascial Closure:

Choosing the appropriate suture:

Choosing the appropriate suture for a given situation requires little more than a basic understanding of the materials available, their merits, and their disadvantages. The effect of an inappropriate choice can be considerable, resulting in unnecessary wound infection, draining sinuses, incisional hernia, or dehiscence.

The ―ideal suture‖ should:

(1) have sufficient strength and maintain it until wound healing is complete,

(2) then disappear so as not to promote patient discomfort or suture granulomas,

(3) have a low index of infectivity and reactivity so as not to promote wound infection and inflammation,

(4) be easily handled and knot securely with minimal difficulty.

Such a suture does not exist. Yet the disadvantages of any one material may be minimized if it is used appropriately.

For many years, the standard suture for fascial closure was **alloy steel wire.** Its tensile strength was incomparable, and as a monofilament, its physical structure minimized foreign body reactivity, bacterial adherence and subsequent wound infections. These attributes were

recognized by Jones et al., who in 1941 reported a tenfold decrease in the incidence of dehiscence (11% to 1.2%) and a pronounced reduction in the incidence of wound infections (27.5% to 0.85%) when mass interrupted alloy steel rather than layered continuous chromic catgut was used for fascial closure[41]. Alloy steel, while preferable to catgut, is not without problems. Most surgeons find it difficult to handle, and its propensity for cutting through gloves is well established[42]. Tight knots are difficult to achieve, and kinking may lead to fracture[32,41]. In addition, its permanent nature may lead to palpable, uncomfortable knots or chronic suture sinuses that eventually require extraction[32,41,42].

It is generally accepted that healing of the midline aponeurosis, with a return of strength comparable to that of intact fascia, requires from 60 to 120 days[43]. Herein lies the problem with **catgut**. Because of its rapid absorption, this material contributes little, if anything, to wound strength. Catgut begins to weaken as early as 5 to 10 days postoperatively[44,45]. Tagart has demonstrated that alter 5 days, in vivo catgut retains only 30% of its original strength and thereafter its support will be completely unreliable[44]. The result is a wound that is unsupported and prone to disruption when even minor stresses are placed on it. The disruption rate has been well documented in numerous reviews of fascial closure with

catgut[41,42,44,46]. Consequently, this material has largely been abandoned for fascial closure.

In light of the above, research during the past years has focused on the development of suture material that embodies the attributes of steel (nonporous with lasting tensile strength and low infectivity) while avoiding the poor handling characteristics and tendency toward sinus formation of steel. Both **monofilament nylon** and **polypropylene (Prolene)** have been developed as desirable substitutes. In surgical use, their inert, nonabsorbable nature has demonstrated lasting strength and a low incidence of dehiscence when used appropriately for abdominal closure.

Hermann, in 1974, first advocated the use of polypropylene after 250 consecutive mass closures with the material without a single dehiscence[47]. Later, Knight and Griffen reported 1,000 consecutive abdominal wound closures with polypropylene (including appendectomies) with an incidence of dehiscence and incisional hernia of only 0.4% and 0.7%, respectively[48]. Comparable results have been achieved with monofilament nylon. The most dramatic of these was the report by Jenkins of 1,505 consecutive continuous, mass closures with nylon with only one failure, an incidence of 0.07%[49].23 Martyak and Curtis reported 280 consecutive midline wounds closed using monofilament nylon and continuous mass closure without dehiscence or incisional herniation[50].

Finally, in two prospective, randomized clinical trials, Leaper et a1 and Pollock et al clearly demonstrated the equivalence of monofilament nylon and stainless steel in achieving minimal dehiscence rates of 0.56% and 0%, respectively[32]. On the basis of these and other clinical trials, polypropylene and monofilament nylon have largely supplanted the use of stainless steel in situations where the latter might be indicated, i.e., the patient at risk for dehiscence.

In general, however, most surgeons remain reluctant to use these sutures routinely, and their reasoning is not unjustified. While more easily handled than steel, both materials have perpetuated one of its lesser qualities—the propensity for creating prolonged incisional discomfort and suture granuloma or sinus formation. Despite ingenious methods bury the knot, reports of these complications, particularly in the thin patient with minimal subcutaneous fat, are numerous[2,32,47,48].

Postlethwait et al, using histologic specimens of suture removed from human tissue at reoperation, demonstrated the minimal reaction elicited by permanent monofliament material in comparison to its multifilament counterpart, silk[40]. While the monofilament was simply encapsulated by a fine zone of fibrous tissue, the multifilament silk evoked a multicellular reaction involving fibroblasts, giant cells, and lymphocytes, surrounding the suture and within the interstices of the multiple filaments. The reason for this inflammatory response is apparent when one considers the structure of silk.

More recently, scanning electron micrographs have been used by Bucknall to demonstrate the intense inflammatory reaction elicited by multifilament silk and other materials in both infected and noninfected states[51]. Katz et al, using radiolabeled bacteria, have quantitated in vitro bacterial adherence to different monofilament and multifilament sutures[52]. They found that monofilament nylon bound the least bacteria while braided sutures (silk, polyglycolic acid [Dexon, Tycron]) had bacterial adherence values fivefold to eightfold higher. After implanting similar bacteria-coated sutures in mice, they found that the inflammatory response or ―degree of infection‖ observed with the various sutures closely correlated with the adherence characteristics noted in vitro. Monofilament nylon, even in the presence of bacteria, consistently evoked a minimal, if even detectable, inflammatory response. All of these observations give credence to the long-held concept that synthetic, monofilament sutures, because of their structure and inert chemistry, are relatively nonreactive and facilitate removal rather than the harboring of bacterial organisms within the wound[76].

Yet the fact that monofilament nonabsorbable sutures can result in chronic sinuses is well established; this is evidently more due to their permanence within the wound than to physicochemical properties. More recent research has therefore focused on the development of long-term

absorbable sutures, which theoretically support the wound long enough for adequate healing and are then absorbed.

Both **polyglycolic acid (Dexon)**, and **polyglactin (Vicryl)** were introduced in the early 1970s, and each has been the subject of extensive clinical trials since that time. Early concerns regarding the use of these materials in abdominal wound closure focused on their absorption and associated declining tensile strength profiles. Both materials are degraded by hydrolysis, but while Vicryl is uniformly absent after 70 days, the process takes somewhat longer for Dexon, which usually requires 3 to 4 months[53]. Loss of tensile strength, however, is rapid, with Dexon retaining zero of its package strength after only 21 days and Vicryl similarly retaining less than 10% of its original strength after 28 days[53].

Yet despite the theoretical possibility of creating a weak wound susceptible to disruption, the use of Dexon and Vicryl has not led to an increased incidence of dehiscence. Murray and Blaisdell closed 650 consecutive abdominal and thoracic incisions[54], and Bentley et al closed 814 consecutive laparotomy incisions with these sutures, with subsequent dehiscence rates of less than 0.5% in each[55]. It appears unequivocal that these absorbable sutures can provide sufficient short-term strength to avoid early post operative dehiscence.

Several investigators have recently extended the concept of ―wound failure‖ to include incisional hernias, and they have considered these a form of ―late dehiscence.‖ Given their time course of absorption and diminution in strength, one might expect a higher incidence of this complication with absorbable sutures. Bucknall et al, in a prospective trial involving 1,129 major laparotomy wounds, reported a significantly higher incidence of incisional hernias in wounds closed with mass polyglycolic acid as opposed to mass nylon (11.5% vs. 7.2%)[13]. Similarly, Pollock et al, in a prospective trial comparing steel, nylon, and polyglycolic acid, found the highest incidence of incisional herniation (13%) when the polyglycolic acid suture was used.[56] Wissing and associates compared interrupted and continuous closures with nylon, polydioxanone (PDS), and Vicryl.[2] They noted incisional hernias in 16.9% and 20.5%, respectively, of Vicryl closures when they are used in an interrupted or continuous fashion. It appears, on the basis of these early results, that the ―long-term‖ absorbable sutures may simply alter the time course of wound failure such that incisional hernia rather than dehiscence is the eventual outcome. While the former is certainly preferable to dehiscence, it is an unfair exchange for the occasional suture sinus that follows closure with the synthetic, nonabsorbable suture materials.

Finally, the results of several recent trials indicate that these absorbable sutures, despite their original intention, are not immune to the complication of chronic sinus formation. Gammelgaard and Jensen noted this complication in 6.5% and 11.3%, respectively, of Vicryl and Dexon closures[16]. Similarly, Bucknall et al reported an identical incidence of sinus formation (11.5%) when either nylon or polyglycolic acid was used[13]. In summary, these absorbable materials, while potentially safe in uncomplicated cases, may lead to a higher incidence of incisional hernias and may result in suture sinus formation, a problem they were specifically designed to avoid.

At present use of able sutures in patients at high risk for dehiscence is sufficiently controversial so as not to be recommended. While monofilament polypropylene and nylon may on occasion lead to chronic suture sinuses, this problem can be virtually eliminated by an appropriate continuous closure and adequate burying of the knots at both ends of the incision. The result is an inherently strong wound, which should endure a moderately stormy postoperative course without disruption.

Technique:

Many surgeons consider the technique of abdominal wound closure to be the single most important factor in prevention of postoperative dehiscence. The results achieved by many with various innovative

techniques are ample evidence that the technical aspects of this ritual are critical.

(a) Layer-by-layer vs. Mass Closure:

Little more than a decade ago, the first consideration at laparotomy closure would have been meticulous reapproximation of the peritoneum. Numerous clinical trials have established that, contrary to previous thinking, this maneuver is unnecessary and unrelated to secure wound healing[25]. Large peritoneal defects heal rapidly with new serosa formation and without increased adhesions[57]. Conversely, sutures that penetrate the peritoneum elicit a substantial foreign body reaction

leading to excessive adhesions and potential intestinal obstruction[57]. Given the obvious risk and doubtful benefit, a separate peritoneal closure should be avoided. Further, fascial reapproximation should be achieved via a preperitoneal technique in which sutures do not penetrate the peritoneum. The layer-by-layer closure advocated by Halsted, though aesthetically pleasing, fails to impart adequate strength to the wound. This method is considerably more time-consuming, and it also adds significantly to the amount of foreign material within the wound, neither of which will benefit the patient.

The evolution of ―mass‖ closure began with the figure-of-eight mass stitch developed by Smead. This method, described by Jones et al, in

a subsequent clinical trial, has since been referred to as the ―Smead-Jones far-and-near technique‖[41]. Strikingly impressive results were responsible for the technique's early popularity. Jones et al reported a tenfold decrement in the incidence of wound disruption (11% to 1.2%) when this method rather than the traditional layered closure was employed[41].

In a more recent study from India by Sivam et al[58], the early and late results of the Smead-Jones (SJ) technique of closure of emergency vertical midline laparotomies was compared with other conventional methods of closure such as anatomical repair (AR), mass closure (MC) and single layer (SL) closure. It was seen that the overall infection rate for SJ at 12.4% was significantly less than all other types of closure. The wound dehiscence rate for SJ at 3.0% was the lowest. This protective effect of SJ against dehiscence was also seen in the presence of post operative chest infection and abdominal distension. The incisional hernia rate for SJ was also lowest (4%). The appearance of the scar was comparable to the other techniques of follow up. This study concluded that the Smead-Jones techniques of laparotomy closure had very low incidence of early and late complications and was superior to other conventional methods of closure. In a similar study by Baggish et al[59], a prospective study of 900 laparotomies utilizing polyglycolic acid suture material and the Smead-Jones closure technique was carried out over a period of 1 year with a reduction in the incidence of wound disruption from 0.4 to 0.1%.

Numerous prospective clinical trials using both the far-and-near as well as the simple mass closure have shown that mass fascial closure results in fewer dehiscences[10,30,49].

(b) Interrupted vs. Continuous Sutures:

Traditionally, the interrupted mass closure using non-absorbable sutures has been used for wounds prone to dehiscence. The trend in recent years has been toward an increased use of the continuous suture. Its advocates cite several advantages over the interrupted method. Chief among these is a comparable, if not slightly lower, incidence of wound disruption when the former was used. The dramatic results reported by Jenkins[49] (1 disruption in 1,505 continuous mass closures) were ample evidence of the security of this technique. He emphasized that large tissue bites, a small stitch interval, and appropriate wound tension were directly responsible for the outcome. When performed correctly, the method uses a length of suture four times as long as the wound. Tissue bite and stitch interval being constant, it is this ratio (suture to wound length) that determines wound tension at closure. The closure allows for potential postoperative abdominal distention without sutures tearing through fascia. This takes advantage of the accepted capability of continuous closures to distribute wound tension along the length of the incision.

Poole et al noted that the continuous technique was not only consistently stronger but that during increasing tension the suture line would often ―shift‖ to accommodate increased stress[9]. In contrast, interrupted closures would rupture suddenly, with initial disruption occurring at the single suture under greatest stress. The authors emphasized that fascial tearing due to wound tension is the primary mediator of incisional dehiscence.

A review by Carlson on acute wound failure emphasized on taking large bites of tissue during closure to prevent dehiscence[60]. Recent randomized prospective clinical trials comparing interrupted and continuous closure have confirmed the security of the latter technique[15,18]. In fact, in the largest prospective trial to date (3,135 patients)[18] the incidence of dehiscence was actually higher (2.0% vs. 0.6%) when interrupted rather than continuous closure was used.

The advantages of a continuous, mass fascial closure include less foreign material in the wound and expediency. The former assumes increasing importance if nonabsorbable suture is used and sinus formation about permanent suture knots is to be avoided. In regard to expediency, most authors have shown that continuous closure reduces operative time by about 20 minutes compared with the interrupted technique[15,50]. While advantageous for any patient, this may be most significant for the unstable

or critically ill patient who is most prone to dehiscence. Given these advantages and the equal, if not greater, wound security provided, the continuous mass closure with non- absorbable suture appears to be the method of choice for wounds at high risk of dehiscence.

RETENTION SUTURES:

Most surgeons employ retention sutures in the presence of multiple risk factors for dehiscence, or when a single risk factor is present in combination with systemic disease sufficient to warrant concern about adequate fascial healing. Irrespective of the cause, the intent of using retention sutures is to hold cut fascial edges in apposition and thereby reduce strain on the incisional suture line until adequate healing has taken place. The ability of retention sutures to perform this function is entirely on how they are placed in relation to the incision.

Based on findings at autopsy, Price[61] demonstrated why ―conventional‖ retention sutures, which simply traversed all layers of the abdominal wall, often failed:

(i) When sutures are place, there is a circumferential distribution of suture tension with minimal support of the fascial aponeurosis.

(ii) With edema on the third day there is increased pressure on the soft tissue.

(iii) Increased suture tension causes pressure injury to tissues in skin and fascial layers at about 7 days. The suture cuts through skin ad fascial layers, reducing lateral support.

(iv) As edema subsides, more fascial support is lost, permitting wound disruption.

Based on the above, Price advocated a retention suture designed primarily to oppose the lateral distracting forces occurring at the incision. (Fig.3, Fig.4) Large sutures traverse the midline to grasp a large bite of the contralateral musculofascial layer; they are then brought back across to the original side, emerging near the point of entry. Consecutive sutures are placed on alternating sides of the wound and anchored either to an overlying frame or to large buttons with tension appropriate to maintain fascial apposition (Fig.5). This was followed by simple interrupted closure of the anterior sheath.

More commonly used retention sutures of today are placed 4 to 5 cm lateral to the incision, traverse all layers of the abdominal wall (with the exception of peritoneum) and cross the midline beneath the mass fascial closure just under the posterior sheath (Fig.6). The following principles have to be adhered to during their placement:

(a) The suture should enter the skin closer to the incision than to the point at which it traverses the ipsilateral posterior fascia. Only then can the fascia

be approximated without compression of the overlying skin and potential necrosis.

(b) A suture that is large enough should be used, no. 2 polypropylene, to avoid the tearing of fascia under considerable tension. The use of sterile intravenous (IV) tubing has been advocated for this purpose.

(c) Peritoneal penetration has to be avoided so as to avoid the increased intra abdominal adhesions provoked by this maneuver which can further lead to small bowel obstruction or fistula.

If abdominal distention is present preoperatively, retention sutures that are taut at the time of closure may become slack during convalescence, leaving the wound susceptible to dramatic, episodic increase in tension as a result of paroxysmal coughing or vomiting. Consequently, they should be checked on a regular basis to ensure that tension is maintained. Depending on the condition of the patient, it is generally safe to remove these sutures between 14 and 21 days postoperatively.

PRESENTATION AND MANAGEMENT OF DEHISCENCE

Many a times wound disruption will occur despite extra preventive measures taken at the initial operation. Early recognition and prompt treatment are critical if the usual mortality of 30% is to be reduced.

In many instances, the time at which dehiscence occurs postoperatively will suggest the cause of the problem. Efron[62] and Lehman and

Partington[30] have pointed out that wound disruptions occurring before the fifth postoperative day cannot be attributed to poor wound healing. These are usually the result of some technical error. This explanation accounts for a minority of dehiscences.

The most frequent interval for dehiscence is between the seventh and eighth postoperative days[3,4,6,30,62], frequently following an episode of severe coughing or vomiting, or progressive abdominal distention secondary to ileus[4,5,26,30,62]. Its development under such circumstances may be all too obvious and accompanied by eventration—the ―burst abdomen.‖ At other times, the only indication of a problem may be a profuse, pink, serosanguinous incisional discharge that leads to the removal of skin sutures revealing viscera in the wound. The pink serosanguinous drainage is associated with dehiscence so often that it is wise to take the patient to the operating room, do a sterile preparation of the abdomen, drape off the wound, and then explore the wound. If a dehiscence is found, the patient can be anesthetized and the wound can be closed with minimal peritoneal contamination.

Irrespective of the presentation of dehiscence, once the diagnosis is confirmed, the **principles of initial management** are as follows:

(1) If eventration has taken place, intestines are replaced in the peritoneal cavity and covered with warm, saline-soaked dressings. Frequent moistening of dressings will prevent the desiccation of involved bowel and will minimize heat and evaporative fluid losses.

(2) A nasogastric tube is passed both for intestinal decompression purposes and to empty the stomach in preparation for general anesthesia.

(3) Intravenous fluids are resumed at a rate that considers both maintenance requirements and the additional losses due to drainage of peritoneal fluid and evaporation from exposed bowel.

(4) After wound cultures have been obtained, broad-spectrum antibiotic therapy is initiated.

Abdominal wound dehiscence is clearly a surgical emergency. Yet this complication rarely necessitates that the patient be taken immediately to the operating room. The resuscitative measures outlined above are initiated to ensure that the patient is properly prepared for reoperation and that additional risks, primarily related to anesthesia, are avoided. Consequently, electrolytes and hemoglobin should be quickly determined, particularly in patients with known derangements, so that corrective measures may be instituted before reoperation.

Surgical Management:

1. Once the patient is fully anesthetized, the wound is reopened along its entire length and the fascial suture line is inspected to determine the cause of dehiscence. Slipped knots, broken sutures, and fascial tears are noted. The fascia is carefully inspected to ensure that a necrotizing fascial infection has not set in. All residual suture material and necrotic or devitalized tissue are removed.

2. At this point formal exploratory laparotomy is performed. A diligent search for intraabdominal abscesses is carried out, particularly if the patient's clinical course

has been consistent with ongoing intraabdominal infection. Once identified, these are drained through separate wounds in the abdominal wall. If dehiscence has taken place during the course of an otherwise uneventful recovery, relaparotomy provides an opportunity for simple visual inspection of enteric anastomoses to assure that subsequent recovery is imminent. In the absence of symptoms consistent with intestinal obstruction, aggressive lysis of small bowel adhesions is not advocated, because these will only reform with greater vigor, thereby increasing the probability of subsequent obstruction.

3. Before closure, the entire abdomen is irrigated with several liters of a warm saline-antibiotic solution (commonly bacitracin or a cephalosporin). This is particularly important following evacuation of an intraabdominal abscess. Although the ability of peritoneal lavage to inhibit the reformation of intraabdominal abscesses is theoretical, its favorable impact on subsequent wound infection has been substantiated[63].

4. Fascial closure is achieved, following principles outlined previously. Evidence suggests that continuous reclosure using heavy nonabsorbable suture material (0 polypropylene), with large tissue bites (1.5 cm), a small stitch interval, and appropriate wound tension works best. Retention sutures should be used in this setting. Both these and the continuous suture line should be placed via a preperitoneal technique.

5. Finally, the wound is irrigated with an antibiotic- saline solution. In all but extremely obese patients, a loose approximation of subcutaneous tissue with absorbable suture may be performed. Primary skin closure is to be avoided. According to work by Mendoza et al[28], the wound infection rate doubled (30% to 60%), following primary skin closure after disruptions. The subcutaneous level is packed open with saline-moistened gauze dressings.

6. Postoperatively, the wound is managed with twice-daily dressing changes to debride accumulating necrotic material and promote formation

of healthy granulation tissue. The decision to close the skin after 5 to 7 days of such treatment depends on the surgeon's preference but requires sound clinical judgement. Meissner and Meiser[36] reported no objective differences between wounds that were closed primarily and healed by primary intention and those managed openly as above. The incidence of wound infection following delayed primary closure is roughly 10%.

7. Additional important postoperative measures include continued nasogastric decompression until intestinal function has returned. Nutritional support is maintained during this period of prolonged postoperative ileus. Consideration should be given to central venous hyperalimentation at this time if not initiated already. Finally, deep breathing exercises via the hand-held spirometer will assist in removal of tracheobronchial secretions and minimize postoperative coughing with its detrimental effect on wound healing. A continuous epidural narcotic infusion to control pain might also be a consideration.

8. In the rare event that a massive necrotizing fascial infection is present, one must adopt a course that requires wide debridement of all involved fascia muscle and soft tissue. The resulting defect is frequently large and defies routine reclosure. If no closure is accomplished, massive dehiscence is certain. In these cases a large sheet of polypropylene mesh is cut to

overlap the defect margins by 1.5 inches. A plane is developed between the peritoneum and the deepest fascia. Using 2-0

monofilament or braided coated synthetic suture the mesh is sutured superficial to the peritoneal surface and deep to the fascia. The outer edge of the mesh is folded over about 0.25 inches, and the cut edge is on the outside so only a smooth mesh surface is on the bowel side. The interrupted sutures are placed at intervals of 0.75 inches with both ends going through the full thickness of the structures, subcutaneous tissues, and the skin. They are tied through large plastic buttons. The mesh can be tailored as the sutures are placed and tied. The abdomen can be washed through the resulting ―screen door‖. Wet antibiotic-saline dressings are applied. If systemic infection is controlled and no fistulas are present, there is a good chance of success. Granulation tissue develops through the mesh and final coverage is achieved with meshed skin grafts with an expansion ratio of 1.5:1.

A novel method of closure of a gaping abdominal wound was described by Tripathy et al, where they used a radial artery pedicle flap to cover exposed mesh which had been used to cover a gaping abdominal wound[64] (Fig.7, Fig.8)

SURGICAL TECHNIQUES FOR PLANNED RELAPAROTOMY:

Planned reexploration of the abdomen following dehiscence is indicated in circumstances like advanced peritonitis and ischemic bowel necrosis. Re-exploration has been advocated to repeatedly clear the abdominal cavity of pus and necrotic debris by warm irrigation and debridement. It is also possible at such a reoperation to confirm the viability of questionably revascularized bowel.

Staged Relaparotomy (STAR)

The most experience with planned reexploration has been obtained with the relaparotomy technique of Whittmann et al[65]. Etappenlavage is a series of planned multiple operative procedures performed at 24-hour intervals. These stepwise procedures were conceived to treat patients at high risk for diffuse advanced purulent peritonitis. The goal of these procedures is to carefully remove as much purulent material as possible and allow the abdomen to be left open enough to prevent tension or increased abdominal pressure. The abdomen is temporally closed using one of four techniques:

(a) retention sutures (R-TAC)

(b) ordinary zippe (Z-TAC)

(c) plastic slide fastener or Glider (G-TAC), or

(d) adhesive alloplastic sheets (Velcro equivalent) (V-TAC)

Of the four different methods employed, R-TAC and Z-TAC are associated with the greatest incidence of complications because of the inability to release the intraabdominal pressure, which resulted in severe necrosis of the abdominal wall. G-TAC provided adequate intraabdominal decompression, but it often opened, thus introducing more pathogens into the abdominal cavity. These problems were avoided by using V-TAC, which accomplished both decreasing intraabdominal pressure and prevented visceral contents from escaping. The etappenlavage procedure for diffuse peritonitis arguably ensures improved elimination of the infectious source, better reduction of bacterial inoculum, and better elimination of toxic necrotic material (Fig.9).

Laparoscopic Methods:

In a study by Eypasch et al, a laparoscopic indwelling cannula was inserted at the time of initial laparotomy for peritonitis or bowel necrosis. The goal of this procedure was to facilitate a relatively atraumatic reexploration and preinsertion of a cannula during the primary operation; this was presumed to greatly decrease the risk of bowel injury by Veress needle[66]. The technique consisted of placing a 15-cm-long and 12-mm-diameter disposable cannula opposite the site of main interest in the abdomen. The internal end was buried in a pocket of rectus muscle made through an additional peritoneal incision. The external portion of the

cannula was sutured to the skin, and on laparoscopic reexploration the suture was cut, the cannula removed from the rectus pocket, and the abdomen insufflated to 15 mm Hg. The study showed that no incisional hernia resulted because insufflating the abdomen was well tolerated by the abdominal wall. No survivor needed relaparotomy. The main drawback was that pain was a problem in conscious patients because movement was restricted as a result of the implantation of the cannula. In the future, a more flexible cannula may facilitate this intriguing strategy of laparoscopic reexploration.

Prognosis:

The incidence of incisional hernia following relaparotomy for dehiscence is high, with most large series reporting rates upto 30%[4,42]. Similarly, the reported mortality rate associated with dehiscence remains at 30%. The encouraging fact is that most deaths associated with dehiscence today are the result of ongoing primary disease rather than being secondary to peritonitis as a direct result of this complication. In the absence of associated progressive disease, patients experiencing an uncomplicated dehiscence have an excellent prognosis.

MATERIALS AND METHODS

Source of Data:

After obtaining approval from the Hospital Ethical Committee (Ref. No. 15806/E4/3/2010, copy enclosed in Annexure), patients admitted to Tirunelveli medical college hospital between april 2017 and October 2018 diagnosed with dehiscence of abdominal wound after undergoing surgical intervention in TVMCH were included in the study.

Type of study **:** Retrospective study

Sample size **:** 80 patients

Inclusion criteria:

All patients with the clinical diagnosis of abdominal wound dehiscence, as evidenced by separation of layers of the abdominal wall postoperatively.

Exclusion criteria:

All patients undergoing gynaecological procedures and relaparotomy

Data collection:

Data regarding following aspects were collected:

- Age
- Gender
- Clinical presentation
- Type of surgery undergone (Elective/ Emergency)

- Presence of contributing factors -

1. Infection (local/systemic)
2. Anaemia (defined as blood haemoglobin <13g/dL in males and <12g/dL in females)
3. Hypoproteinaemia (defined as serum total protein <6g/dL)
4. Postoperative cough or vomiting
5. Uremia (defined as serum urea >40mg/dL and/or serum creatinine >1.4mg/dL)
6. Electrolyte abnormalities (normal serum sodium 135 - 145mEq/L, normal serum potassium 3.5 - 5mEq/L)
7. Ascites
8. Surgical technique (type of incision, suture material used for abdominal closure)
9. Obesity (defined as Body Mass Index >30kg/m^2)
10. Comorbid conditions if any
11. Drug use if any

- Management
- Outcome

Statistical analysis:

Standard clinical and statistical methods were employed to analyze the data.

RESULTS

AGE DISTRIBUTION

Table 2: Age Distribution

Age	No. of cases	Percentage
15-30	8	10
31-45	22	25
46-60	42	52.5
>60	16	12.5

Maximum cases (52.5%) were found to be in the 46-60 years age group (Graph 1). The youngest patient in this study was 15 years old and the oldest was 74 years.

SEX DISTRIBUTION

Table 3: Sex Distribution

Sex	No. of cases	Percentage
Male	55	68.75
Female	25	31.25

There was a marked male predominance in the sex distribution (68.75%)

PRIMARY DISEASE

Table 4: Primary Disease

Primary Disease (Diagnosis)	No. of cases	Percentage
Gastrointestinal perforation (including trauma)	30	37.5
Biliary pathology	7	8.75
Malignancy	4	5
Intestinal gangrene	5	6.25
Others	28	35

Patients included in the study had been operated on for diverse surgical conditions, most common among the study population being perforated duodenal ulcer (37.5%). Next common conditions were ileal perforation (10%) and paraumbilical hernia. Out of 80 patients with wound dehiscence, 30 (37.5%) had gastrointestinal perforation (including traumatic perforations), while 7 patients (8.75%) had some form of surgery of the biliary tract. 4 patients (5%) were operated for some intraabdominal malignancy.

SURGERY UNDERGONE

Table 5: Surgery Undergone

Surgery	No. of cases	Percentage
Graham's omental patch closure	31	38.75
Primary closure of ileal perforation	9	11.25
On lay mesh repair for paraumbilical hernia	18	22.5
Open cholecystectomy	7	8.5
Resection and anastomosis	8	10
Ileostomy	2	2.5

The most common surgery that preceded the onset of abdominal wound dehiscence was found to be laparotomy with Graham's omental patch closure for perforated duodenal ulcer (25%). The other common surgeries undergone were laparotomy with primary closure of ileal perforation (20%) (25% of whom underwent an ileotransverse anastomosis also) and laparotomy and drainage of ruptured liver abscess (7.5%). 12.5% of the surgeries involved some form of bowel anastomoses and 7.5% of surgeries in the study population involved creation of colostomy.

NATURE OF SURGERY

Table 6: Nature of surgery (elective or emergency)

Nature	No. of cases	Percentage
Elective	18	22.5
Emergency	62	77.5

77.5% cases of abdominal wound dehiscence were found to occur following emergency surgery and 22.5% following elective surgery (Graph 2).

PREOPERATIVE RISK FACTORS

I.PREVALENCE OF ANEMIA

Table 7: Prevalence of Anemia

Anemia	No. of cases	Percentage
Present	58	72.5
Absent	22	27.5

Thus the prevalence of anemia among the study population was found to be 72.5% (Graph 3).

II. PREVALENCE OF HYPOPROTEINEMIA

Table 8: Prevalence of Hypoproteinemia

Hypoproteinemia	No. of cases	Percentage
Present	49	61.25
Absent	31	38.75

Hypoproteinemia was noted in 61.25% of cases (49 patients). (Graph 4).

III. LONG TERM DRUG USE

Table 10: Long term drug use

Drug	No. of cases	Percentage
Antihypertensives	2	2.5
Insulin	10	12.5
Eltroxin	4	5
None	64	80

Most patients (80%) had no history of any concomitant drug intake. 2.5% were on antihypertensive medication while 12.5% were on insulin and 5% on eltroxin. No patients had history of chronic steroid use.

IV. PREVALENCE OF OBESITY

Table 11: Prevalence of Obesity

Obesity	No. of cases	Percentage
Present	28	35
Absent	52	65

35% of the study population had a BMI of >30kg/m^2 while 65% were non-obese.

Table 12: PREOPERATIVE RISK FACTORS - SUMMARY

CAUSE	No. of cases	Percentage
Anemia	22	27.5
Hypoproteinemia	49	61.25
Obesity	28	35
Diabetes mellitus	14	17.5
Steroids	-	-

CLINICAL PRESENTATION

Table 13: Clinical presentation of dehiscence

Symptom	No. of cases	Percentage
Pus discharge (PD)	42	52.5
Serous discharge (SD)	28	35
Wound gaping (WG)	10	12.5

52.5% of cases presented with pus discharge from the wound around 6-8th post operative day prior to developing wound dehiscence. 35% presented with serous discharge from the wound (Fig.10), while 12.5% presented with painless gaping of the operative wound (Graph 5).

TIME OF DISRUPTION

Table 14: Time of Presentation

Postoperative day	No. of cases	Percentage
4	8	10
5	6	7.5
6	8	10
7	28	35
8	30	37.5

Most cases were found to present with burst abdomen on the 7^{th} and 8^{th} postoperative days (35% and 37.5% respectively), with the highest incidence on the 8^{th} postoperative day (Graph 6).

INCISION

Table 15: Incision used

Incision	No. of cases	Percentage
Midline	34	42.5
Transverse	18	18
McBurney's	21	26.25
Right subcostal	7	8.75

Midline incision was found to be the most common incision used in the preceding surgery in the study population (42.5%), the next common one being McBurney's (26.25%) (Graph 7)

POSTOPERATIVE RISK FACTORS

I. POSTOPERATIVE COUGH

Table 17: Prevalence of Post operative Cough

Post operative cough	No. of cases	Percentage
Present	52	65
Absent	28	35

Persistent cough in the postoperative period, prior to the onset of wound disruption, was seen in 52 patients (65%).

II. POSTOPERATIVE INFECTION (LOCAL/SYSTEMIC)

Table 18: Prevalence of Post operative Infection

Post operative Infection	No. of cases	Percentage
Present	66	82.5
Absent	14	17.5

Infection in the post operative period, in the form of either localized wound infection or septicemia, was noted in 82.5% of the cases (66 cases). (Graph 9)

III. OTHER POSTOPERATIVE COMPLICATIONS (IF ANY)

Table 19: POSTOPERATIVE RISK FACTORS - SUMMARY

CAUSE	NO. OF CASES
Infection	66
Cough	52
Electrolyte imbalance	20
Vomiting	16
Uremia	12
Abdominal distention	10
Bowel leakage	4
Ascites	4

Many post-operative predisposing factors are responsible for burst abdomen. In this study, wound infection (66 cases) and cough (52 cases) were leading factors in the majority of the cases. It is noted that most of the patients had more than one predisposing factor responsible for the development of burst abdomen.

MANAGEMENT

Table 20: Management of Dehiscence

Management	No. of cases	Percentage
Immediate resuturing	16	20
Delayed resuturing	45	56.25
Conservative	19	23.75

76.25% of the cases were managed with secondary suturing using non absorbable suture material, of which 20% were subjected to immediate resuturing, while 56.25% cases had delayed resuturing after adequate control of wound infection and ingrowth of granulation tissue. 23.75% (19 patients) were managed conservatively due to poor surgical risk from coexisting septicemia and multiorgan failure (Graph 9).

DISCUSSION

Abdominal wound dehiscence is one of the most dramatic and serious post operative complications after any major abdominal surgery. Acute wound failure can present as mechanical wound separation or dehiscence. Dermal wound separation worsens cosmetic results but is unlikely to cause significant harm, while abdominal wall wound failure can have life-threatening outcomes. Irrespective of the presentation of dehiscence, once the diagnosis is confirmed, the initial management includes replacement of intestinal contents into the peritoneal cavity and covering with moist saline packs, gastric decompression with nasogastric tube, intravenous fluids and broad spectrum antibiotics. Though it is considered a surgical emergency, the patient should be stabilized and any antecedent cause that led to dehiscence, if reversible, be corrected before embarking on surgical treatment. Surgery for burst abdomen involves reopening and inspecting the entire surgical wound, exploratory laparotomy to look for any intraabdominal abscesses or anastomotic leaks, thorough peritoneal lavage, and a good reclosure (continuous reclosure using heavy nonabsorbable suture material such as 0 poly propylene, with large tissue bites of 1.5 cm, a small stitch interval, and appropriate wound tension works best) along with application of retention sutures.

In this study involving 80 patients who developed abdominal wound dehiscence postoperatively, most (62%) of patients had undergone a prior

emergency laparotomy. This observation is in comparison with that done by Penninckx et al[27] who reported a 76% prevalence of emergency laparotomy in a study group with dehiscence.

In the present study, the mean age where the maximum cases were clustered was 46-60 years (42 cases, 52.5%).

Male predominance was noted in this study, with 87.5% of the study population being males and 12.5% being females. Thus male:female ratio was 7:1. Hampton[21] observed that males are three times more often affected than females (1963).

A detailed analysis of various factors which impede wound healing was done, taking into consideration the factors that existed preoperatively and those that resulted from the primary condition that warranted surgery, or the surgery itself. Important among the preoperative factors is anemia which leads to reduced capillary perfusion, which in turn results in a low tissue oxygen tension, causing collagen defects and impaired wound healing.58 out of 80 patients in the present study (72.5%) were found to be anemic. At least 70% of the normal hemoglobin level is required for elective safe surgery. Joergenson and Smith also noticed in their study a higher incidence of abdomen wound dehiscence in patients having anemia[69].

The prevalence of hypoproteinemia in the study population was 61.25%. This observation is comparable to reports by Wolff[3], Alexander

and Pavdden[20] and Keill et al[25] that 62%, 71% and 85% of their respective wound dehiscences were associated with hypoproteinemia. Every effort should be made to correct these nutrient deficiencies in the preoperative period before planned surgery.

The role of chronic comorbidities in causing wound disruption was also studied. Important among them is diabetes mellitus. The clean wound infection rate is higher in diabetic patients (11%) than in the general patient population[70]. A convincing result could not be arrived at from the present study due to its retrospective nature, and since only one patient in the study group was diabetic.

Long term use of pharmacological agents like chemotherapeutic drugs and steroids has also been proven to cause wound disruption[60]. Corticosteroid use delays epidermal repair but there is no solid evidence that myofascial or GI healing is impaired.

Another important predisposing factor is obesity. Bucknall et al[13] described a higher risk of wound failure in obese patients owing to increased intraabdominal pressure, reduced respiratory reserve, higher rate of pulmonary complications, and a greater infection rate in adipose tissue. 20% of the study subjects who developed dehiscence were obese.

The most frequent interval at which dehiscence occurred in this study group were the 7^{th} and 8^{th} postoperative days (35% and 37.5% respectively). This was comparable to the results obtained by Wolff[3], Guiney

et al[4], Greenburg et al[6], Lehman et al[30], Efron et al[62], Hampton et al[21], Alexander et al[20] and Keill et al[25]. A study by White et al also showed that disruption most commonly occurs during the second postoperative week[67].

The most prevalent presenting feature in the study group was purulent discharge from the wound (21 cases, 52.5%). This may imply localized wound infection as an impediment to wound healing that led to acute wound failure. Similar observations were made by DuBay et al[71]. The classic description of a profuse serosanguinous discharge preceding the onset of dehiscence was noted in 14 patients (35%) in the present study.

In this study, 42.5% of wound dehiscence occurred in vertical midline incisions, with the remaining patients had right subcostal (8.75%), transverse (22.5%) or McBurney's (26.25%) incisions. Parmar and Gohil et al[72] describe various factors which hold midline incision at a higher risk of dehiscence than other incisions.

Even with good patient selection and good surgical technique, wound dehiscence cannot be totally avoided as a host of postoperative events have a vital role to play in wound healing. The most important is postoperative infection which leads to sloughing out of the stitches and separates the rectus sheath. Wound infection is more common in emergency operations and patients presenting with peritonitis. In the present study, 82.5% of patients had evidence of infection, either limited to

the wound or systemic. Fleischer et al[74] noted that deep wound infection was a clear risk factor for dehiscence.

Post-operative cough also leads to high frequency of abdominal wound dehiscence. 65% of patients in this study had persistent cough in the postoperative period prior to the onset of dehiscence. Wolff reported severe paroxysmal coughing prior to wound disruption in over 60% of cases[3].

Post-operative abdominal distention and vomiting lead to vigorous tension on the suture line and breaking up of stitches, leading to burst abdomen. Bowel leakage leads to peritonitis and infects the wound. 5% of study subjects had bowel leak, either from the anastomotic site or from the closure site of a perforation, as evidenced by draining of bowel contents through the intraperitoneal drain in the postoperative period. In the post-operative period, uremia, electrolyte imbalance and ascites also have an effect on wound healing. Based on various risk stratifications, a risk model for predicting the chance of dehiscence has been proposed by van Ramshorst[73].

In this study, 16 out of 80 patients were treated by immediate resuturing of the wound. 45 patients, initially treated conservatively by daily dressings, underwent delayed resuturing of wound after adequate control of local infection and ingrowth of granulation tissue.19 out of 80

patients were treated conservatively as they were not fit for surgery, in the form of daily dressings.

CONCLUSION

Wound dehiscence is a serious sequel of impaired wound healing. It occurs most commonly above the age of 50 years, predominantly in males and with vertical midline abdominal incisions. Many factors can predispose to this grave complication. Knowledge of the more common mechanisms and how to avoid or overcome these hazards help to reduce the incidence of this dangerous complication. The more common factors contributing to wound disruption can be summarized as follows:

Presence of pre-operative anemia, hypoproteinemia, and cough favor high incidence of burst abdomen. Emergency surgery precludes adequate patient preparation and correction of preexisting abnormalities, and hence forms an independent risk factor. During operation, peritoneal contamination, improper choice of suture material and poor suturing technique predispose to burst abdomen. Post-operatively, unusual abdominal wall strain from persistent cough, vomiting, abdominal distention, uncontrolled wound infection, ascites and bowel leakage attribute to the development of burst abdomen.

Prompt and early diagnosis of abdominal wound dehiscence and proper treatment decrease morbidity and mortality. If the above predisposing factors are well understood before doing any abdominal surgery, the present incidence and mortality rates can be reduced further.

Abdominal wound dehiscence is as old as surgery. Predisposing factors are either patient or surgeon related. Despite several incisions and suture materials, controversy remains, with no consensus on the ideal methods or materials for closure of abdominal wounds to prevent dehiscence. At best, the incidence of dehiscence can be reduced.

FIGURE – 1

LAYERS OF ABDOMINAL WALL

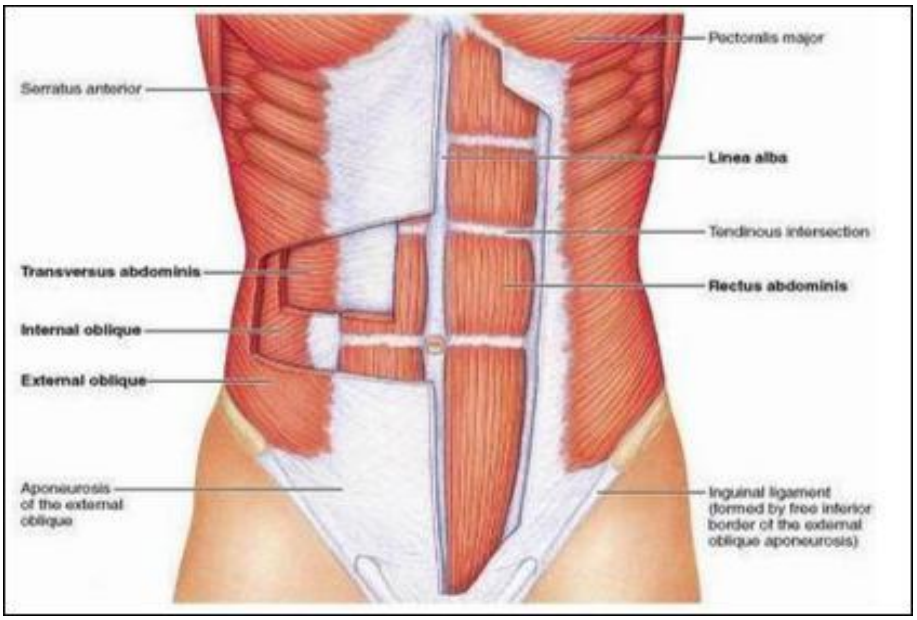


FIGURE – 2 RELATIONSHIP OF MUSCULAR APONEUROSES ABOVE AND BELOW SEMICIRCULAR LINE

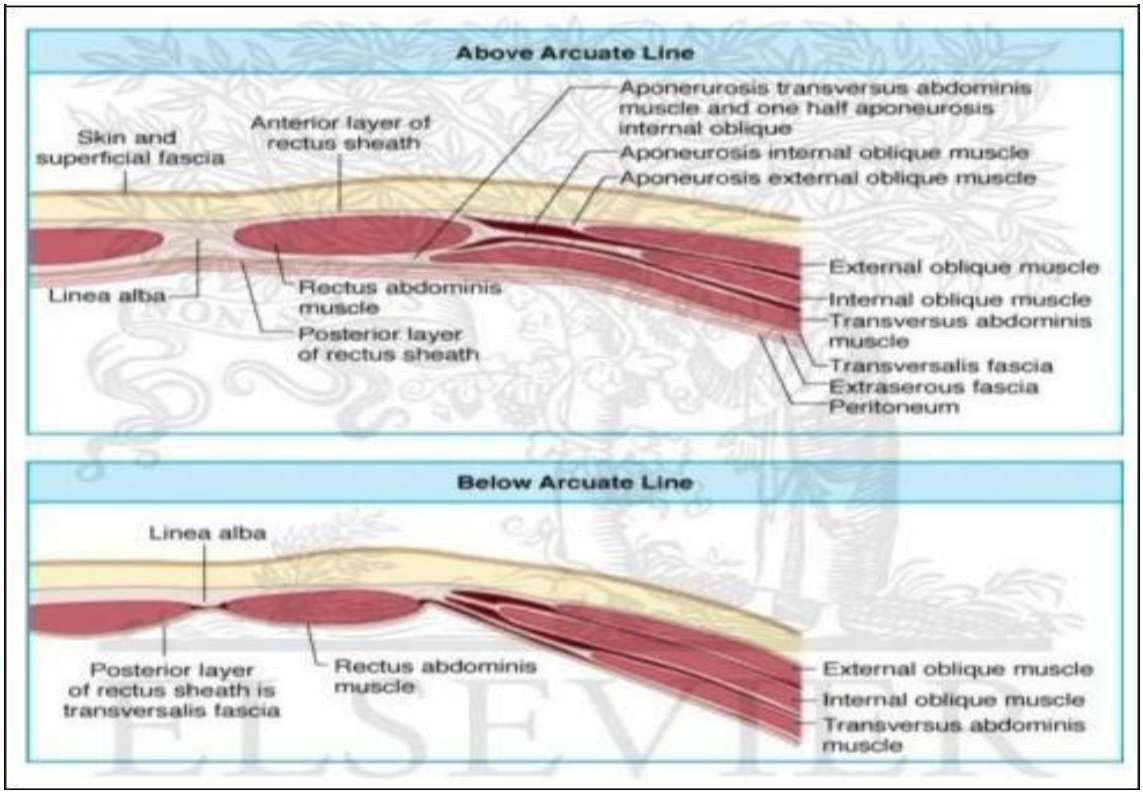


FIGURE – 3
PRICE'S RETENTION SUTURES

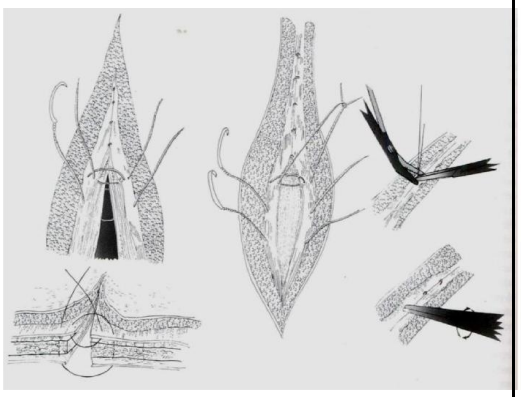


FIGURE – 4

PRICE'S STITCH

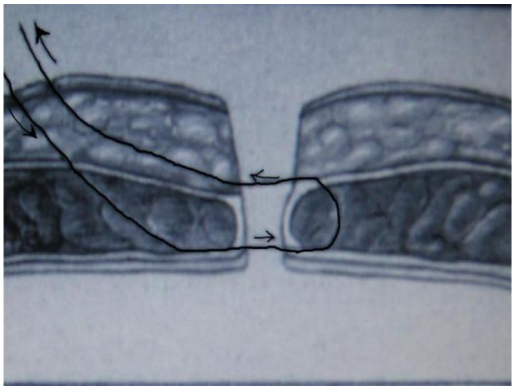


FIGURE – 5

ANCHORING OF RETENTION SUTURES USING (A) FRAME, (B) BUTTONS

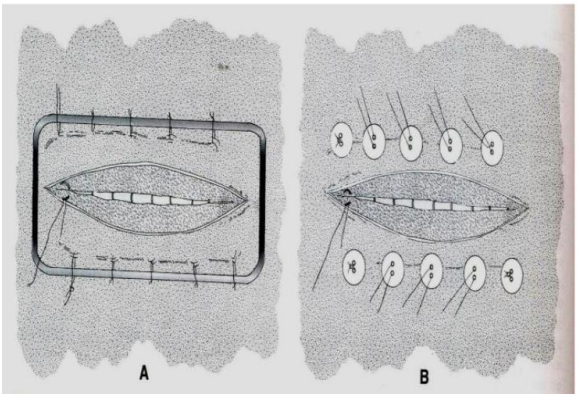


FIGURE – 6

CONVENTIONAL RETENTION SUTURES

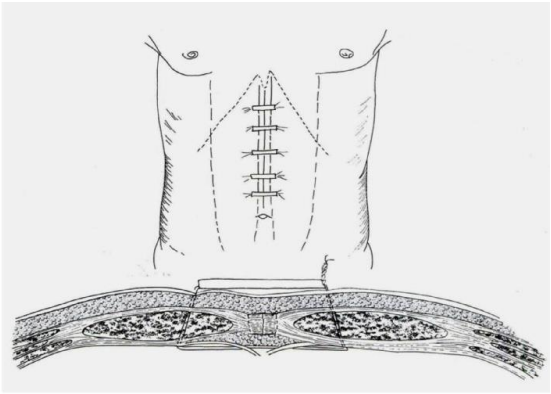


FIGURE - 7

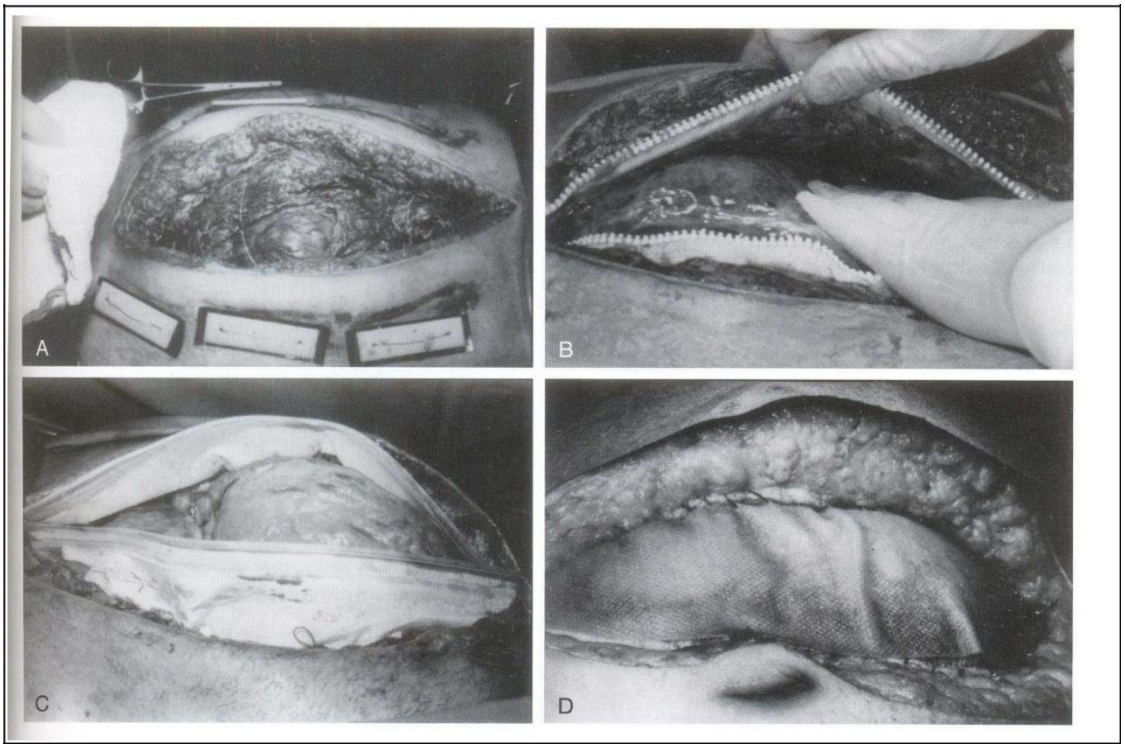
CLOSURE OF GAPING ABDOMINAL WOUND USING RADIAL ARTERY PEDICLE FLAP

FIGURE - 8

FINAL RESULT AFTER CLOSURE WITH RADIAL ARTERY PEDICLE FLAP

FIGURE - 9

DIFFERENT METHODS FOR TEMPORARY ABDOMINAL CLOSURE



(A) ETAPPENLAVAGE USING RETENTION SUTURES (R-TAC)
(B) B) ETAPPENLAVAGE USING ZIPPER (Z-TAC)
C) ETAPPENLAVAGE USING SLIDE FASTENER (G-TAC)
D) ETAPPENLAVAGE USING ADHESIVE VELCRO SHEETS (V-TAC)

FIGURE - 10

SEROSANGUINOUS DISCHARGE FROM WOUND ON 6TH POD

FIGURE – 11

SAME WOUND ON 8TH POD SHOWING DEHISCENCE

FIGURE – 12

WOUND DEHISCENCE WITH WITH EXPOSED SMALL BOWEL

FIGURE – 13

WOUND DEHISCENCE IN A PATIENT

WITH END COLOSTOMY

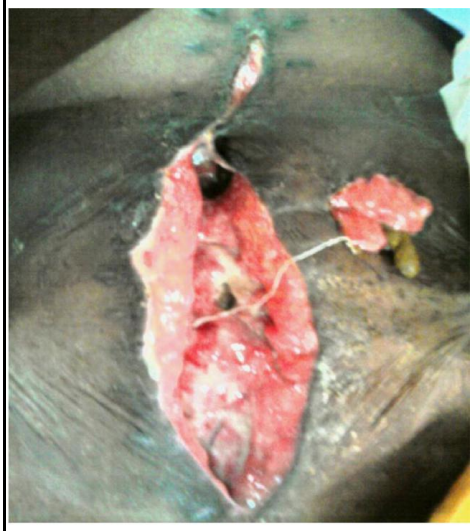


FIGURE – 14

RESUTURING WITH RETENTION SUTURES (HORIZONTAL TENSION SUTURE) (HORIZONTAL MATTRESS

FIGURE – 15

RESUTURING WITH RETENTION SUTURES (VERTICAL TENSION SUTURE)

FIGURE – 16

HEALED WOUND 14 DAYS AFTER RESUTURING

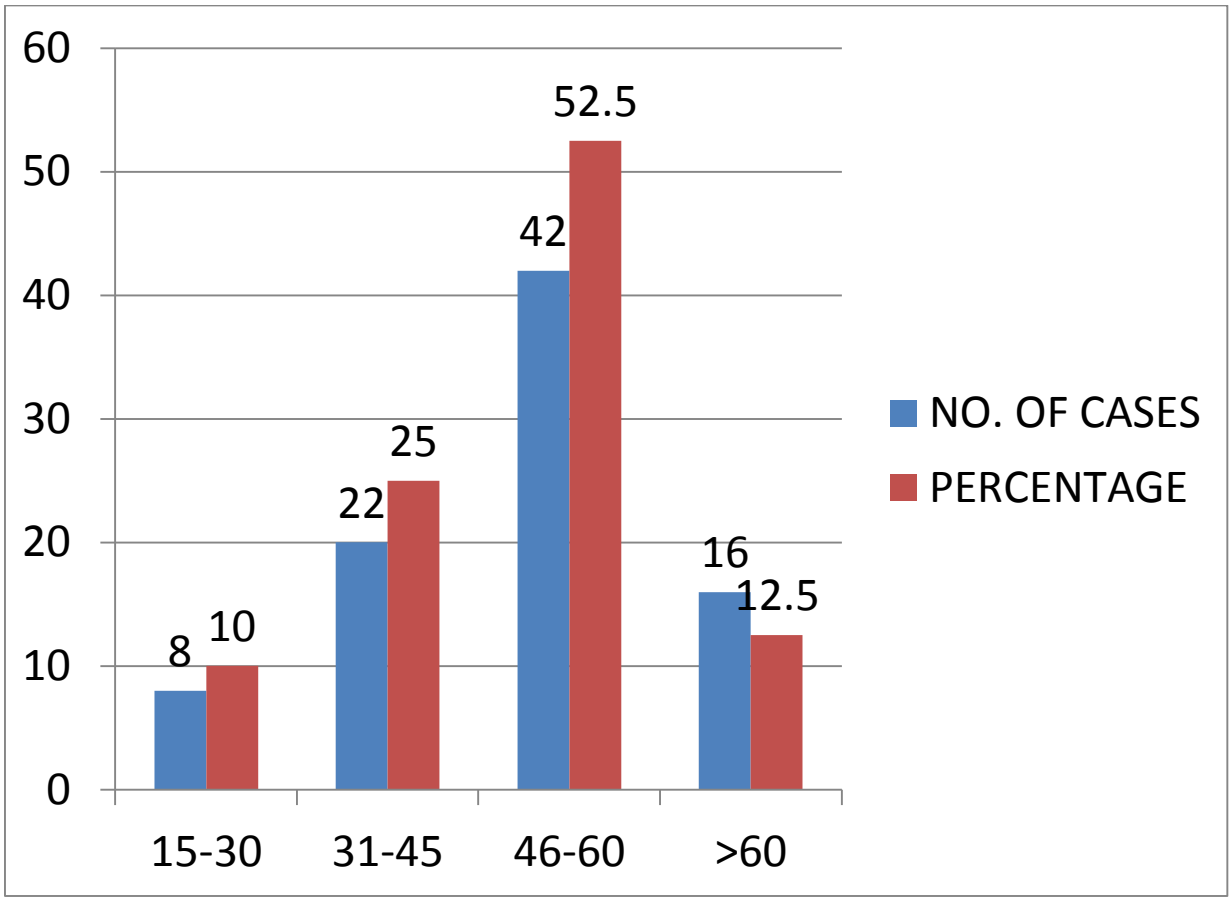
FIGURE – 17

GAPING WOUND WITH HEALING BY SECONDARY INTENTION

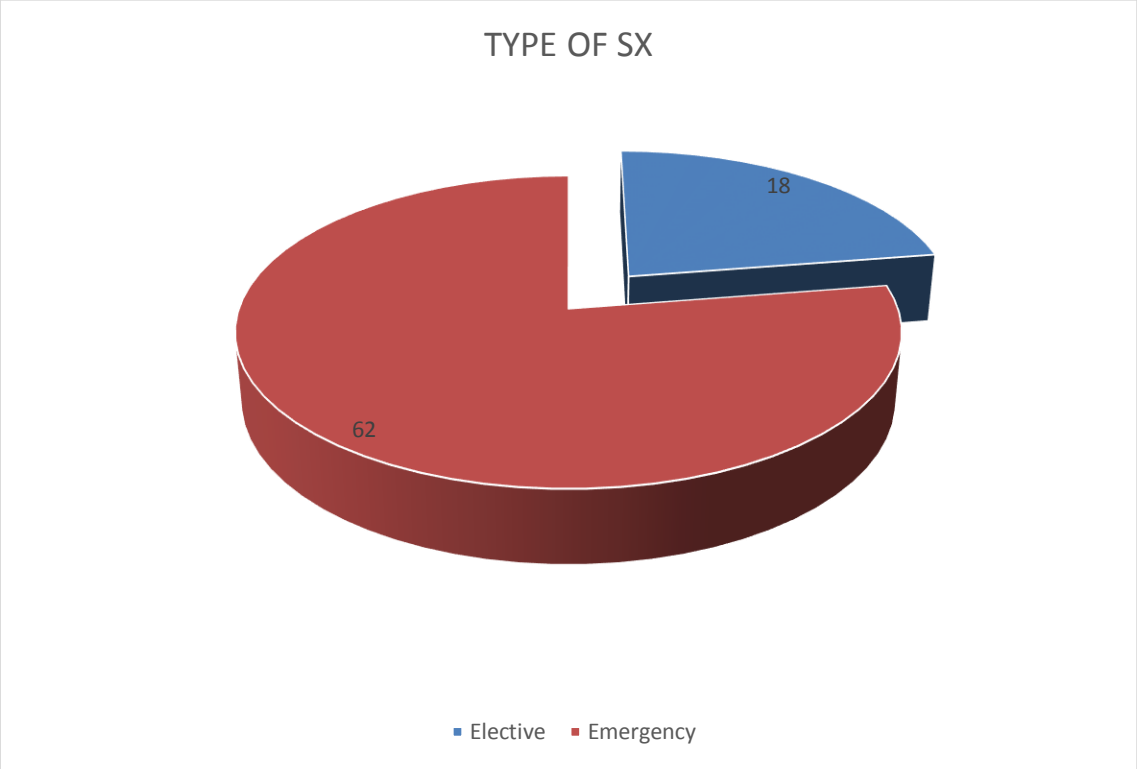
RESULTS AND OBSERVATIONS

AGE DISTRIBUTION

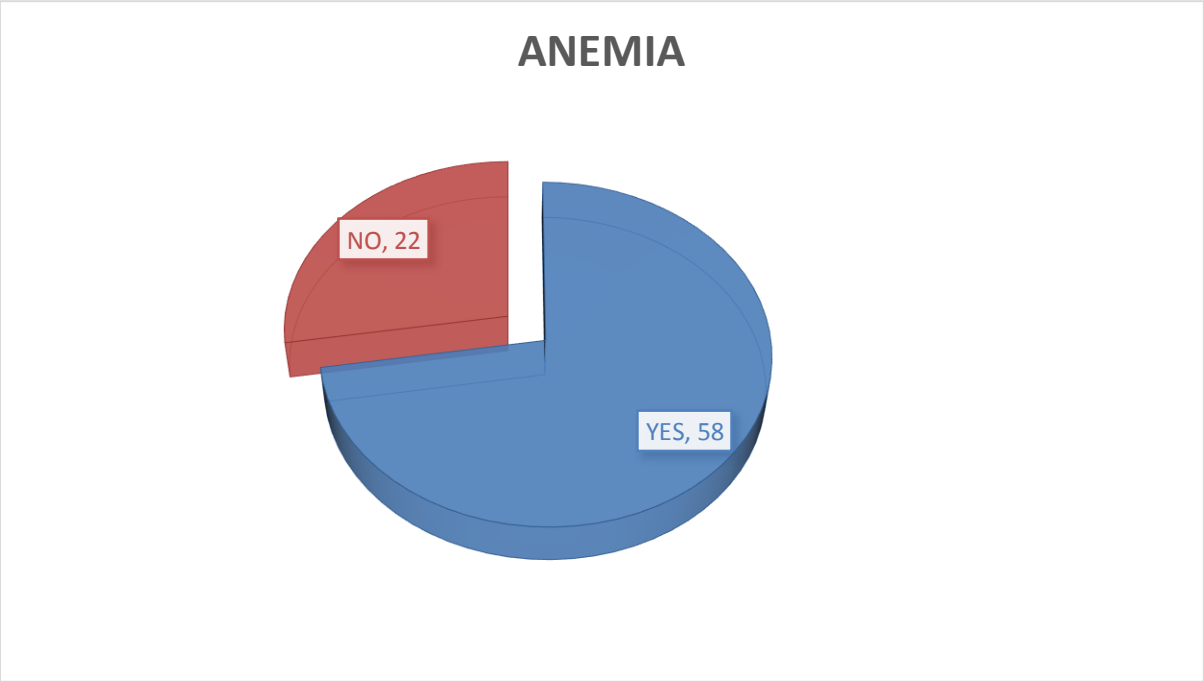
GRAPH-1



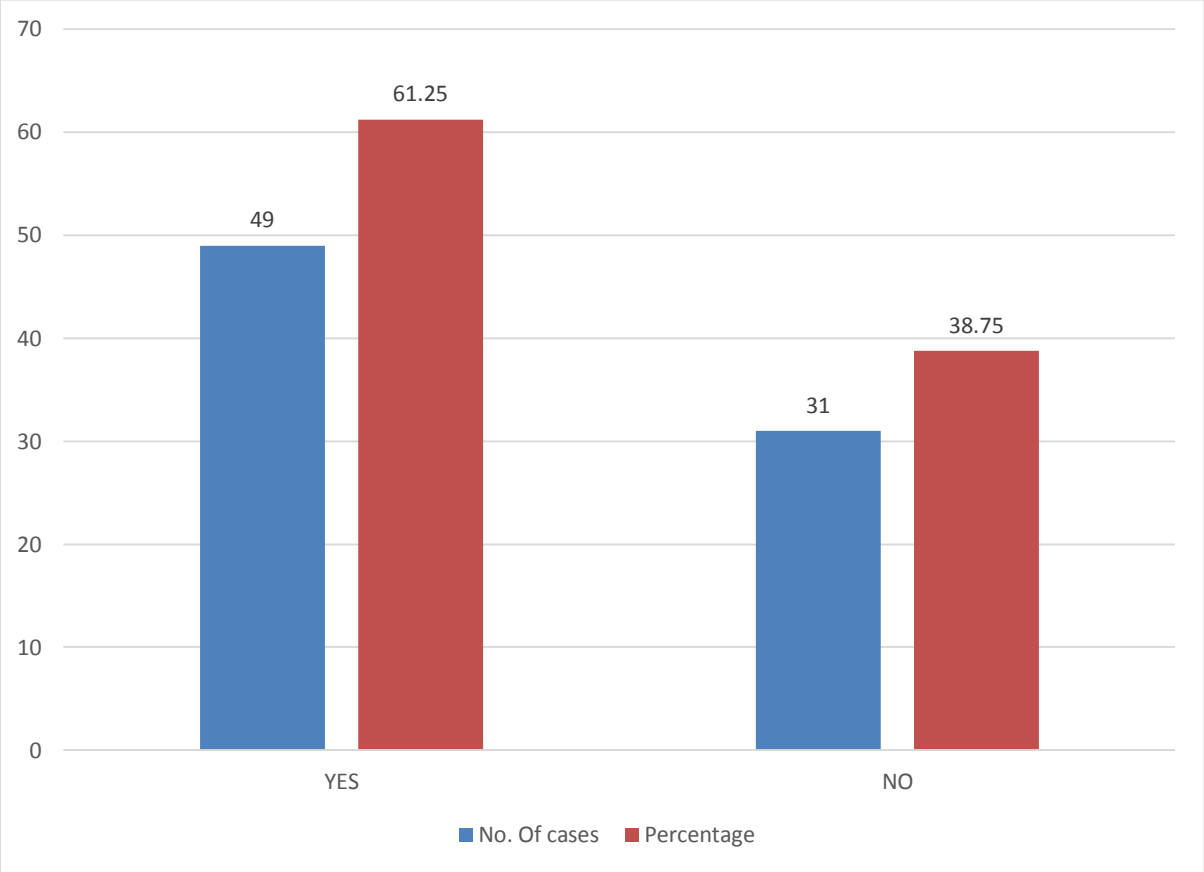
GRAPH 2- NATURE OF SURGERY



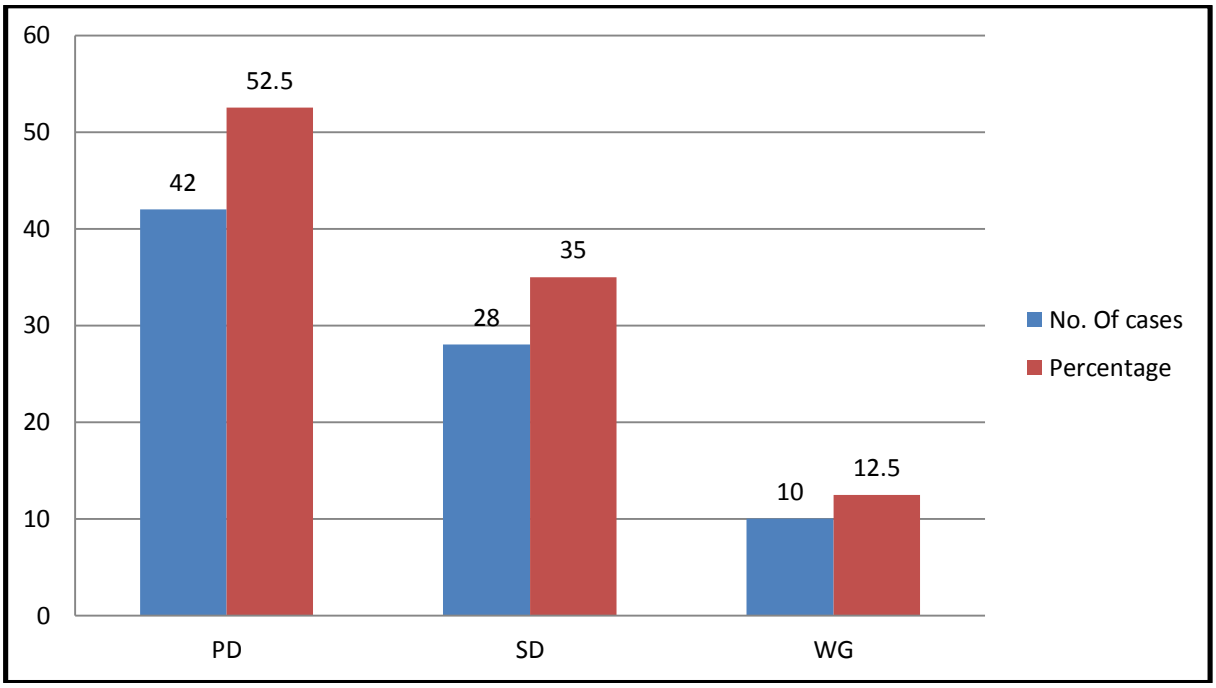
GRAPH 3PREVALANCE OF ANEMIA



GRAPH 4 PREVALANCE OF HYPOPROTEINEMIA

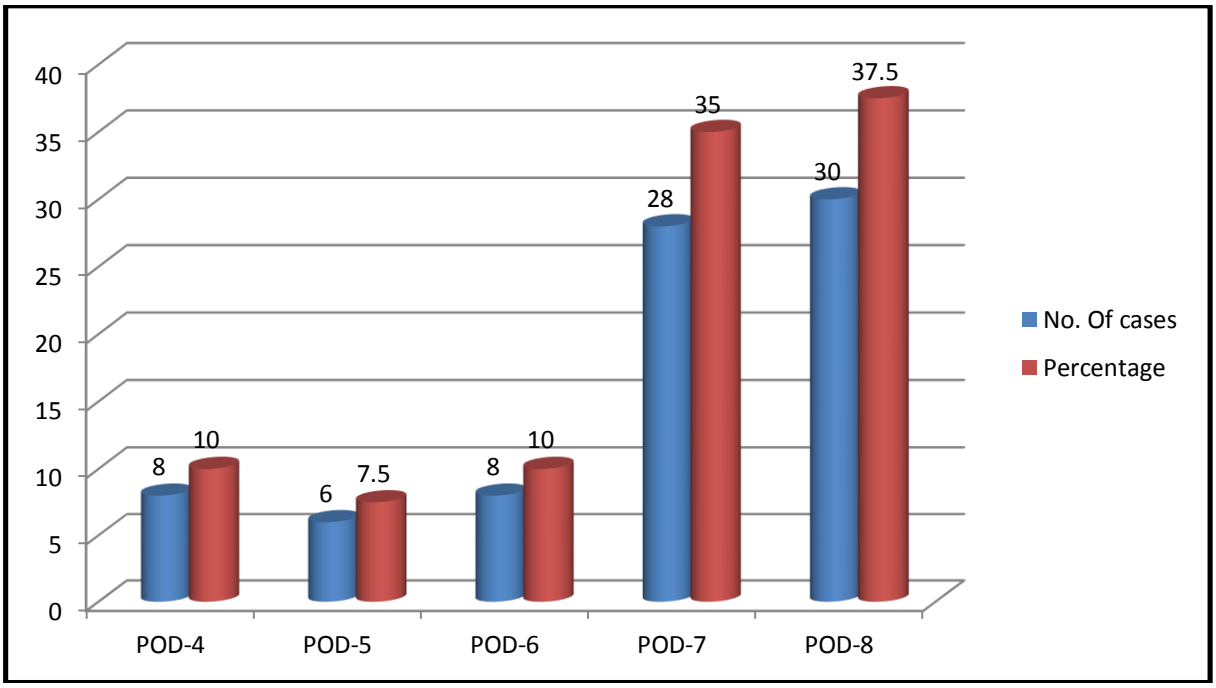


GRAPH 5 ‘CLINICAL PRESENTATION

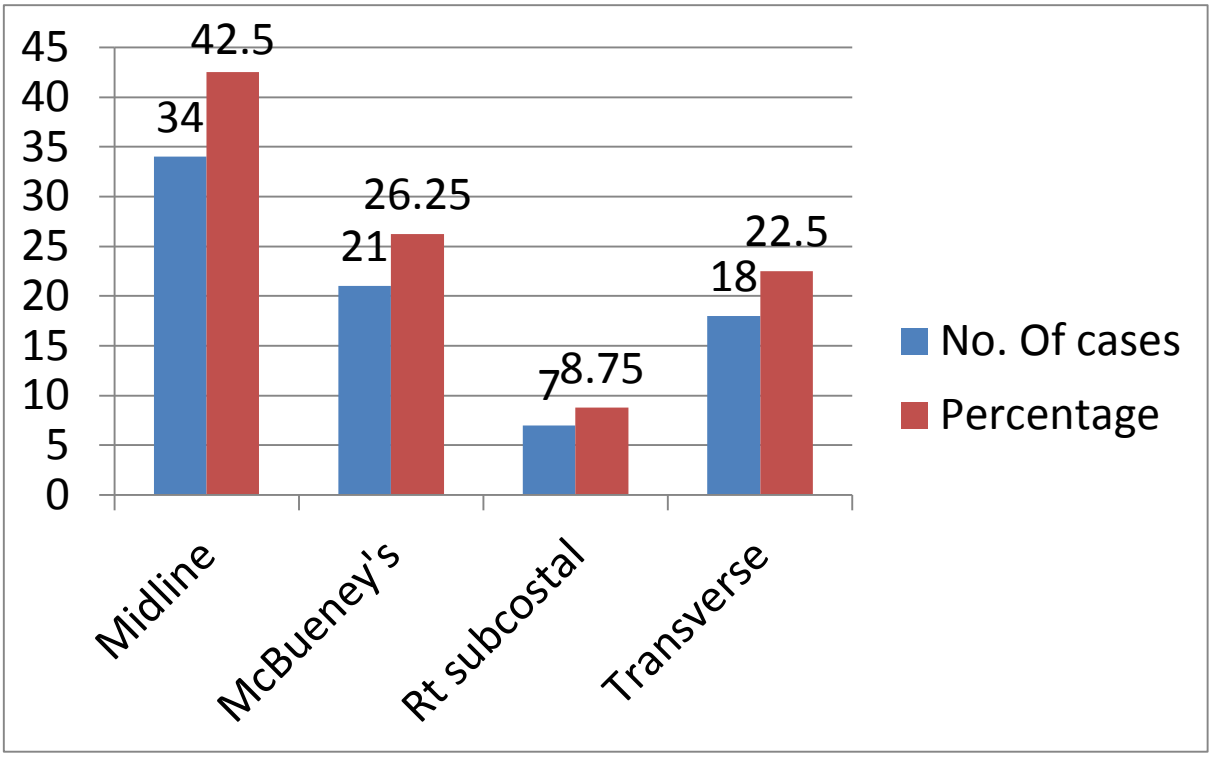


PD-Purulent discharge ; SD-Serous discharge; WG-Wound gaping

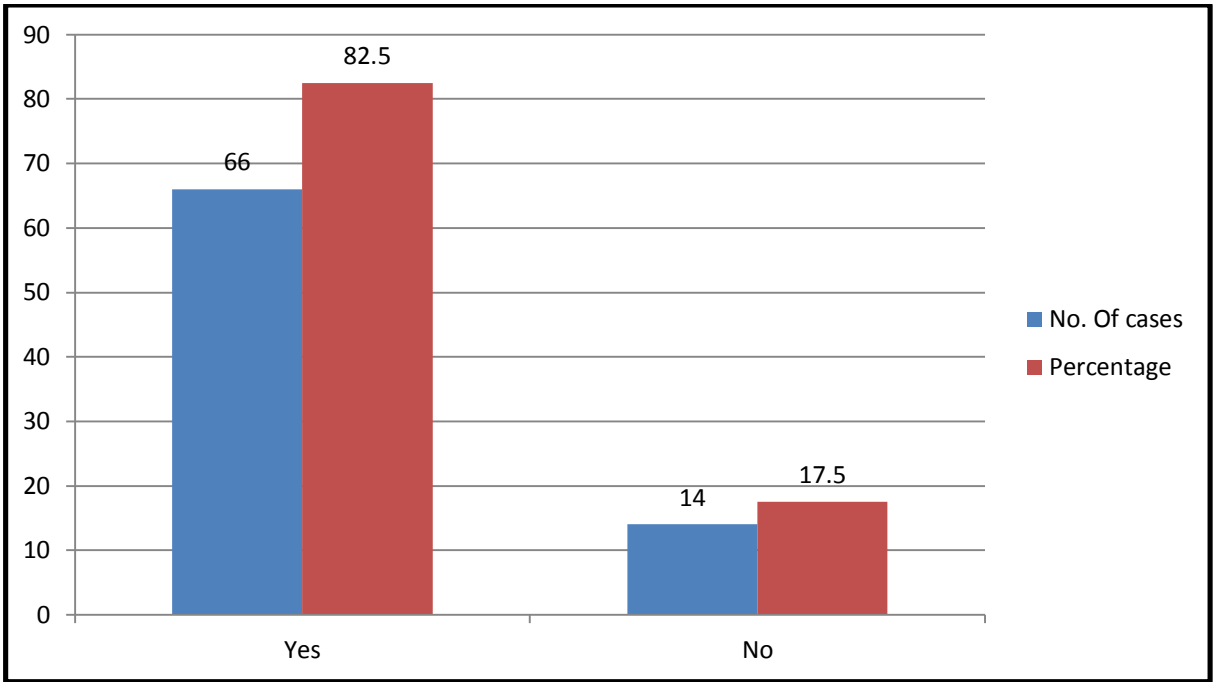
GRAPH 6 TIME OF PRESENTATION



GRAPH 7 TYPE OF INCISION

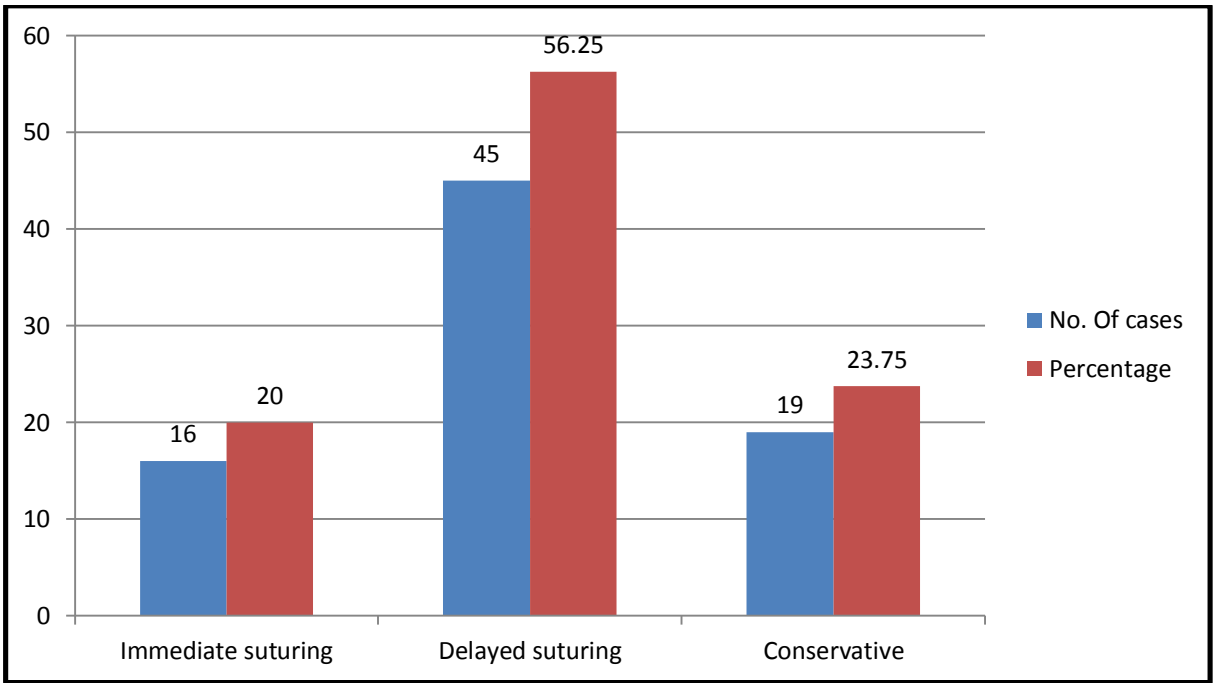


GRAPH 8 POST OP INFECTION



GRAPH 9

TYPE OF MANAGEMENT



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ANNEXURES

PROFORMA

Name:

Age:

Sex:

IP No.

Diagnosis:

HISTORY:

A. Surgery done:

B. Whether elective or emergency:

C. Clinical presentation of dehiscence:

a) burst on ___ post operative day

b) presented with serous discharge/purulent discharge/wound gaping

c) History of cough in post operative period:

d) History of vomiting in postoperative period:

D. Other comorbidities, if any:

E. Drug use if any:

EXAMINATION:

A. General Examination:

1) Height:

Weight:

Body Mass Index (BMI) - [weight in kg/(height in metres)^2] :

2) Vitals:

Pulse rate:

Blood pressure:

Respiratory rate:

3) Temperature:

4) Pallor: +/-

5) Pedal edema: +/-

6) Nail changes (if any): B. Abdominal Examination:

1) Inspection:

a. Wound: presence and nature of discharge, changes in surrounding skin

b. Abdominal distention: +/-

c. Enterostomy: +/-

2) Palpation:

a. Warmth

b. Tenderness

c. Guarding/rigidity

d. Organomegaly

3) Percussion: free fluid in abdomen +/-

4) Auscultation:

5) Nature of output from intraperitoneal drain:

C. Cardiovascular examination: heart sounds, murmurs if any

D. Respiratory system:

a. Air entry:

b. Breath sounds:

c. Added sounds if any

INVESTIGATIONS:

a. Haemoglobin-

b. Total count, differential count-

c. Serum urea-

d. Serum creatinine-

e. Serum electrolytes (sodium, potassium, chloride)-

f. Serum proteins-

TECHNICAL DETAILS OF SURGERY:

a) Type of incision -

b) Whether enterostomy (colostomy/ileostomy) done or not-

c) Suture material used for abdominal closure -

d) Technique used for closure-

MANAGEMENT GIVEN FOR WOUND DEHISCENCE:

Surgical/Conservative

If conservative, reason for opting non-surgical management:

OUTCOME:

SL.No	NAME	AGE	SEX	IP.No	DIAGNOSIS	SURGERY	NATURE OF SURGERY (EL/EM)	CLINICAL PRESENTATION	PRESENTATION ON POD	POSTOPERATIVE COUGH (Y/N)	POST OPERATIVE INFECTION (LOCAL/SYSTEMIC)	COMORBIDITIES
1	ALAGULAKSHMI	43	F	45394	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	PD	5	Y	Y	A,HP,O
2	VELUSAMY	65	M	41339	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	PD	7	Y	Y	A,O
3	RAMASAMY	78	M	41360	INT.OBS.WITH ILEAL GANGRENE	RESECTION ANASTAMOSIS	EM	SD	6	N	Y	A
4	MARIAMMAL	44	F	45403	INT OBSTRUCTION	BAND RELEASE	EM	SD	8	N	N	OA
5	MURUGAN	51	M	41370	ILEAL PERFORATION	PRIMARY CLOSURE	EM	PD	4	N	Y	A,HP
6	MARIAPPAN	29	M	41510	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	SD	8	N	Y	A
7	ANBALAGAN	22	M	41624	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	PD	7	N	N	HP,O
8	LAKSHMI	35	F	45507	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	PD	8	Y	Y	A,HP
9	PANDI	61	M	43269	GALL BLADDER PERFORATION	OPEN CHOLECYSTECTOMY	EM	WG	8	N	N	O,HPA
10	NAGARAJAN	31	M	43219	OBSTUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	SD	7	Y	Y	A,HP

11	CHINNATHAI	54	F	45464	CALCULOUS CHOLECYSTITIS	OPEN CHOLECYSTECTOMY	EL	PD	8	Y	Y	HP,O, A
12	MAHARAJAN	24	M	43328	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	PD	8	Y	Y	A,HP
13	ASHOK KUMAR	47	M	42744	ADVANCED CA STOMACH	FEEDING JEJUNOSTOMY	EL	PD	6	Y	N	A
14	KALYANIKU MAR	23	M	44998	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	WG	4	N	Y	A,HP
15	AKILLA	32	F	45505	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	PD	5	Y	Y	A,HP, O
16	ESSAKIMUTH U	67	M	44985	CA STOMACH	GASTRECTOMY	EL	PD	8	Y	Y	A
17	BALASARAS WATHY	34	F	47253	OBSTRUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	PD	8	N	Y	O,A
18	SELVAM	24	M	45052	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	WG	7	N	Y	A,HP, O
19	CHELLAPPA	68	M	45019	ADHESIVE INTESTINAL OBSTRUCTION	LAPAROTOMY & ADHESIOLYSIS	EM	WG	8	N	Y	HP,A
20	KANNAIAH	60	M	49658	INTESTINAL OBSTRUCTION- CA COLON	ILEOSTOMY		SD	7	Y	Y	A,HP
21	JEYAPRIYA	14	F	47257	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	PD	8	N	Y	A
22	SHANMUGAM	61	M	46749	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	PD	4	Y	Y	A
23	BALRAJ	42	M	46812	ADHESIVE INTESTINAL OBSTRUCTION	LAPAROTOMY & ADHESIOLYSIS	EM	SD	7	Y	Y	HP
24	SABITHA	23	F	47250	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	SD	8	Y	Y	A,O
25	SUNDARAM	69	M	46730	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	SD	7	Y	Y	

26	RANJITH	24	M	46828	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	PD	8	N	Y	A,HP
27	MYDEEN	41	M	46921	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	PD	4	Y	N	HP
28	ANNAPARVATHY	18	F	49073	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	PD	6	Y	Y	A
29	KATHIRESAN	18	M	46910	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	PD	7	Y	Y	A
30	GNANAMANI	70	F	49076	INT.OBS.WITH ILEAL GANGRENE	RESECTION ANASTAMOSIS	EM	WG	7	Y	Y	HP,OA
31	ARUMUGAM	83	M	46802	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	PD	4	N	Y	HP
32	VELUDOSS	64	M	46731	CA STOMACH WITH GOO	ANTERIOR GASTROJEJUNOSTOMY	EL	PD	5	Y	N	A,HP
33	KAVERI	34	F	49195	ILEAL PERFORATION	PRIMARY CLOSURE	EM	PD	7	Y	Y	O
34	GANESN	50	M	46917	OBSTRUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	PD	8	Y	Y	A
35	GEORGE	55	M	47759	INT.OBS.WITH ILEAL GANGRENE	RESECTION ANASTAMOSIS	EM	PD	4	N	Y	O
36	BALAMANI	45	F	49199	CALCULOUS CHOLECYSTITIS	OPEN CHOLECYSTECTOMY	EL	WG	6	N	Y	A,HP,O
37	CHITHIRAIVEL	50	M	48563	RECURRENT APPEDICITIS	OPEN APPENDICECTOMY	EL	PD	8	Y	Y	HP
38	SURESH	18	M	46743	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	PD	7	N	Y	HP
39	PANEER SELVAN	61	M	50539	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	SD	8	Y	N	O
40	JOTHI	19	F	49080	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	PD	5	Y	Y	A
41	MOORTHI	19	M	51326	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	PD	6	Y	Y	A,HP

42	JAMILABEEVI	43	F	50299	OBSTRUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	WG	6	Y	N	HP,OA
43	SUBASH	24	M	52369	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	PD	7	N	Y	O
44	SRINATH	32	M	52798	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	SD	7	N	Y	A,HP
45	PREETHI	17	F	47358	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	SD	8	Y	Y	HP
46	KUMARAN	43	M	52986	ADHESIVE INTESTINAL OBSTRUCTION	LAPAROTOMY & ADHESIOLYSIS	EM	PD	7	Y	Y	A,HP,O
47	RAVIKUMAR	29	M	53654	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	SD	8	Y	Y	A,O
48	ANANTH	38	M	53412	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	WG	7	N	N	A
49	MOOKKAMMAL	57	F	49029	GALL BLADDER PERFORATION	OPEN CHOLECYSTECTOMY	EM	SD	8	N	Y	A,HP.O
50	SUBURAJ	41	M	53918	OBSTRUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	PD	6	N	Y	O
51	KALIMUTHU	29	M	54102	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	SD	7	N	Y	
52	KALIAMMAL	50	F	51211	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	WG	4	N	Y	A
53	VELLUDURAI	43	M	54210	BLUNT TRAUMA ABDOMEN	MESENTERIC TEAR REPAIR	EM	PD	8	N	Y	HP
54	MANIKANDAN	29	M	55321	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	PD	5	Y	N	A,HP,O
55	KARTHIK	36	M	54961	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	PD	7	N	Y	A,HP

56	SELVI	45	F	52838	OBSTRUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	SD	8	N	Y	A
57	MANI	31	M	54927	ILEAL PERFORATION	PRIMARY CLOSURE	EM	WG	8	Y	Y	HP
58	KISHORE	39	M	55601	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	PD	8	N	Y	HP
59	PRAVEEN	21	M	54960	ILEAL PERFORATION	PRIMARY CLOSURE	EM	PD	7	Y	N	A,HP
60	DEVASENA	43	M	54983	ADHESIVE INTESTINAL OBSTRUCTION	LAPAROTOMY & ADHESIOLYSIS	EM	WG	8	N	Y	A,HP
61	MUTHULAKSHMI	36	F	54665	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	SD	8	N	Y	HP
62	SREEJITH	28	M	55634	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	SD	7	N	N	
63	PACKIYAMANI	55	F	54808	CALCULOUS CHOLECYSTITIS	OPEN CHOLECYSTECTOMY	EL	SD	7	Y	Y	A,O
64	ASAIMANI	43	M	53012	OBSTRUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	PD	8	Y	Y	HP,A
65	MOORTHI	51	M	53198	CA ESOPHAGUS	FEEDING GASTROSTOMY	EL	PD	7	N	Y	A,HP
66	VIJAYAN	24	M	54633	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	SD	8	Y	Y	
67	VELLAMANI	70	F	56462	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	PD	8	Y	N	A,HP,O
68	RAJAGOPAL	34	M	55329	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	SD	7	N	Y	O,A
69	SUBRAMANI	56	M	55670	CALCULOUS CHOLECYSTITIS	OPEN CHOLECYSTECTOMY	EL	SD	7	N	Y	A
70	PARVATHY	34	F	56497	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	WG	8	N	Y	HP,O

71	SYED ALI	43	M	56235	OBSTUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	SD	7	N	Y	A,HP
72	JAMES	19	M	56780	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	PD	8	N	Y	A
73	JAVAGAR BEEVI	60	F	56542	ADHESIVE INTESTINAL OBSTRUCTION	LAPAROTOMY & ADHESIOLYSIS	EM	PD	5	N	N	A,HP
74	PREMANAYAGAM	52	M	57901	ADVANCED CA ESOPHAGUS	FEEDING JEJUNOSTOMY	EL	SD	7	Y	YY	HP,A
75	ARAVIND	25	M	56913	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	SD	4	N	Y	A,HP
76	MARIYAMMAL	48	F	56642	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	PD	7	Y	Y	HP
77	MOHAN	39	M	57108	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	SD	7	Y	Y	HP,OA
78	RAM KUMAR	27	M	57209	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	SD	8	N	N	A,HP
79	KRISHNAMMAL	45	F	58224	CALCULOUS CHOLECYSTITIS	OPEN CHOLECYSTECTOMY	EL	SD	7	N	Y	A,O,HP
80	NARAYANAN	57	M	58691	OBSTRUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	SD	6	Y	Y	A,HP

ABBREVIATIONS USED IN MASTERCHART

EL - elective

EM - emergency

POD - post operative day

Y - yes

N - no

perf - perforation

PD - purulent discharge

SD - serous discharge

WG - wound gaping

HP- hypoproteinemia

A-Anemia

O- obesity

SL.No	NAME	AGE	SEX	IP.No	DIAGNOSIS	SURGERY	NATURE OF SURGERY (EL/EM)	CLINICAL PRESENTATION	PRESENTATION ON POD	POSTOPERATIVE COUGH (Y/N)	POST OPERATIVE INFECTION (LOCAL/SYSTEMIC)	COMORBIDITIES
1	ALAGULAKSHMI	43	F	45394	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	PD	5	Y	Y	A,HP,O
2	VELUSAMY	65	M	41339	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	PD	7	Y	Y	A,O
3	RAMASAMY	78	M	41360	INT.OBS.WITH ILEAL GANGRENE	RESECTION ANASTAMOSIS	EM	SD	6	N	Y	A
4	MARIAMMAL	44	F	45403	INT OBSTRUCTION	BAND RELEASE	EM	SD	8	N	N	OA
5	MURUGAN	51	M	41370	ILEAL PERFORATION	PRIMARY CLOSURE	EM	PD	4	N	Y	A,HP
6	MARIAPPAN	29	M	41510	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	SD	8	N	Y	A
7	ANBALAGAN	22	M	41624	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	PD	7	N	N	HP,O
8	LAKSHMI	35	F	45507	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	PD	8	Y	Y	A,HP
9	PANDI	61	M	43269	GALL BLADDER PERFORATION	OPEN CHOLECYSTECTOMY	EM	WG	8	N	N	O,HPA
10	NAGARAJAN	31	M	43219	OBSTUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	SD	7	Y	Y	A,HP
11	CHINNATHAI	54	F	45464	CALCULOUS CHOLECYSTITIS	OPEN CHOLECYSTECTOMY	EL	PD	8	Y	Y	HP,O,A
12	MAHARAJAN	24	M	43328	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	PD	8	Y	Y	A,HP

13	ASHOK KUMAR	47	M	42744	ADVANCED CA STOMACH	FEEDING JEJUNOSTOMY	EL	PD	6	Y	N	A
14	KALYANIKUMAR	23	M	44998	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	WG	4	N	Y	A,HP
15	AKILLA	32	F	45505	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	PD	5	Y	Y	A,HP, O
16	ESSAKIMUTHU	67	M	44985	CA STOMACH	GASTRECTOMY	EL	PD	8	Y	Y	A
17	BALASARASWATHY	34	F	47253	OBSTRUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	PD	8	N	Y	O,A
18	SELVAM	24	M	45052	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	WG	7	N	Y	A,HP, O
19	CHELLAPPA	68	M	45019	ADHESIVE INTESTINAL OBSTRUCTION	LAPAROTOMY & ADHESIOLYSIS	EM	WG	8	N	Y	HP,A
20	KANNAIAH	60	M	49658	INTESTINAL OBSTRUCTION- CA COLON	ILEOSTOMY		SD	7	Y	Y	A,HP
21	JEYAPRIYA	14	F	47257	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	PD	8	N	Y	A
22	SHANMUGAM	61	M	46749	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	PD	4	Y	Y	A
23	BALRAJ	42	M	46812	ADHESIVE INTESTINAL OBSTRUCTION	LAPAROTOMY & ADHESIOLYSIS	EM	SD	7	Y	Y	HP
24	SABITHA	23	F	47250	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	SD	8	Y	Y	A,O
25	SUNDARAM	69	M	46730	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	SD	7	Y	Y	
26	RANJITH	24	M	46828	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	PD	8	N	Y	A,HP
27	MYDEEN	41	M	46921	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	PD	4	Y	N	HP
28	ANNAPARVATHY	18	F	49073	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	PD	6	Y	Y	A

29	KATHIRESAN	18	M	46910	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	PD	7	Y	Y	A
30	GNANAMANI	70	F	49076	INT.OBS.WITH ILEAL GANGRENE	RESECTION ANASTAMOSIS	EM	WG	7	Y	Y	HP,OA
31	ARUMUGAM	83	M	46802	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	PD	4	N	Y	HP
32	VELUDOSS	64	M	46731	CA STOMACH WITH GOO	ANTERIOR GASTROJEJUNOSTOMY	EL	PD	5	Y	N	A,HP
33	KAVERI	34	F	49195	ILEAL PERFORATION	PRIMARY CLOSURE	EM	PD	7	Y	Y	O
34	GANESN	50	M	46917	OBSTRUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	PD	8	Y	Y	A
35	GEORGE	55	M	47759	INT.OBS.WITH ILEAL GANGRENE	RESECTION ANASTAMOSIS	EM	PD	4	N	Y	O
36	BALAMANI	45	F	49199	CALCULOUS CHOLECYSTITIS	OPEN CHOLECYSTECTOMY	EL	WG	6	N	Y	A,HP,O
37	CHITHIRAIVEL	50	M	48563	RECURRENT APPEDICITIS	OPEN APPENDICECTOMY	EL	PD	8	Y	Y	HP
38	SURESH	18	M	46743	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	PD	7	N	Y	HP
39	PANEER SELVAN	61	M	50539	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	SD	8	Y	N	O
40	JOTHI	19	F	49080	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	PD	5	Y	Y	A
41	MOORTHI	19	M	51326	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	PD	6	Y	Y	A,HP
42	JAMILABEEVI	43	F	50299	OBSTRUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	WG	6	Y	N	HP,OA
43	SUBASH	24	M	52369	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	PD	7	N	Y	O
44	SRINATH	32	M	52798	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	SD	7	N	Y	A,HP

45	PREETHI	17	F	47358	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	SD	8	Y	Y	HP
46	KUMARAN	43	M	52986	ADHESIVE INTESTINAL OBSTRUCTION	LAPAROTOMY & ADHESIOLYSIS	EM	PD	7	Y	Y	A,HP, O
47	RAVIKUMAR	29	M	53654	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	SD	8	Y	Y	A,O
48	ANANTH	38	M	53412	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	WG	7	N	N	A
49	MOOKKAMMAL	57	F	49029	GALL BLADDER PERFORATION	OPEN CHOLECYSTECTOMY	EM	SD	8	N	Y	A,HP. O
50	SUBURAJ	41	M	53918	OBSTRUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	PD	6	N	Y	O
51	KALIMUTHU	29	M	54102	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	SD	7	N	Y	
52	KALIAMMAL	50	F	51211	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	WG	4	N	Y	A
53	VELLUDURAI	43	M	54210	BLUNT TRAUMA ABDOMEN	MESENTERIC TEAR REPAIR	EM	PD	8	N	Y	HP
54	MANIKANDAN	29	M	55321	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	PD	5	Y	N	A,HP, O
55	KARTHIK	36	M	54961	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	PD	7	N	Y	A,HP
56	SELVI	45	F	52838	OBSTRUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	SD	8	N	Y	A
57	MANI	31	M	54927	ILEAL PERFORATION	PRIMARY CLOSURE	EM	WG	8	Y	Y	HP
58	KISHORE	39	M	55601	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	PD	8	N	Y	HP
59	PRAVEEN	21	M	54960	ILEAL PERFORATION	PRIMARY CLOSURE	EM	PD	7	Y	N	A,HP
60	DEVASENA	43	M	54983	ADHESIVE INTESTINAL OBSTRUCTION	LAPAROTOMY & ADHESIOLYSIS	EM	WG	8	N	Y	A,HP
61	MUTHULAKSHMI	36	F	54665	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	SD	8	N	Y	HP

62	SREEJITH	28	M	55634	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	SD	7	N	N	
63	PACKIYAMANI	55	F	54808	CALCULOUS CHOLECYSTITIS	OPEN CHOLECYSTECTOMY	EL	SD	7	Y	Y	A,O
64	ASAIMANI	43	M	53012	OBSTRUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	PD	8	Y	Y	HP,A
65	MOORTHI	51	M	53198	CA ESOPHAGUS	FEEDING GASTROSTOMY	EL	PD	7	N	Y	A,HP
66	VIJAYAN	24	M	54633	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	SD	8	Y	Y	
67	VELLAMANI	70	F	56462	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	PD	8	Y	N	A,HP,O
68	RAJAGOPAL	34	M	55329	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	SD	7	N	Y	O,A
69	SUBRAMANI	56	M	55670	CALCULOUS CHOLECYSTITIS	OPEN CHOLECYSTECTOMY	EL	SD	7	N	Y	A
70	PARVATHY	34	F	56497	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	WG	8	N	Y	HP,O
71	SYED ALI	43	M	56235	OBSTUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	SD	7	N	Y	A,HP
72	JAMES	19	M	56780	ACUTE APPENDICITIS	EMERGENCY OPEN APPENDICECTOMY	EM	PD	8	N	Y	A
73	JAVAGAR BEEVI	60	F	56542	ADHESIVE INTESTINAL OBSTRUCTION	LAPAROTOMY & ADHESIOLYSIS	EM	PD	5	N	N	A,HP
74	PREMANAYAGAM	52	M	57901	ADVANCED CA ESOPHAGUS	FEEDING JEJUNOSTOMY	EL	SD	7	Y	YY	HP,A
75	ARAVIND	25	M	56913	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	SD	4	N	Y	A,HP
76	MARIYAMMAL	48	F	56642	APPENDICULAR PERFORATION	EMERGENCY OPEN APPENDICECTOMY	EM	PD	7	Y	Y	HP
77	MOHAN	39	M	57108	PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EL	SD	7	Y	Y	HP,OA

78	RAM KUMAR	27	M	57209	DUODENAL ULCER PERFORATION	GRAHAMS OMENTAL PATCH CLOSURE	EM	SD	8	N	N	A,HP
79	KRISHNAMMAL	45	F	58224	CALCULOUS CHOLECYSTITIS	OPEN CHOLECYSTECTOMY	EL	SD	7	N	Y	A,O,HP
80	NARAYANAN	57	M	58691	OBSTRUCTED PARAUMBILICAL HERNIA	MESH REPAIR ONLAY	EM	SD	6	Y	Y	A,HP